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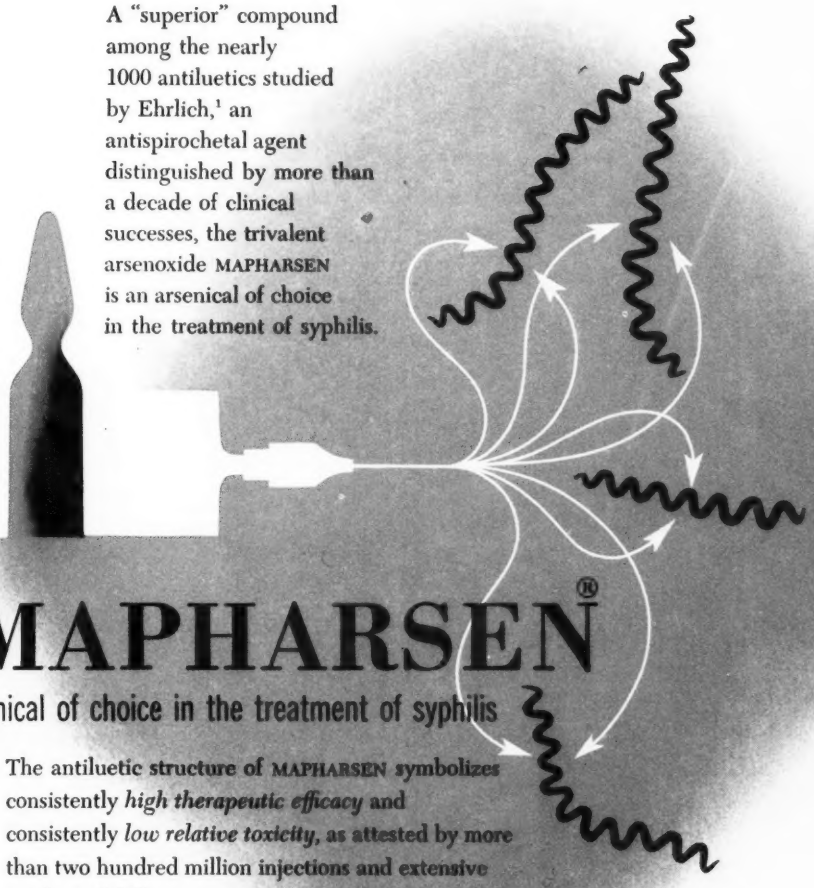

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[1] Krantz, J. C., Jr., and Carr, C. J.: Pharmacologic Principles of Medical Practice, Williams & Wilkins Co., Baltimore, 1949, pps. 114-119.

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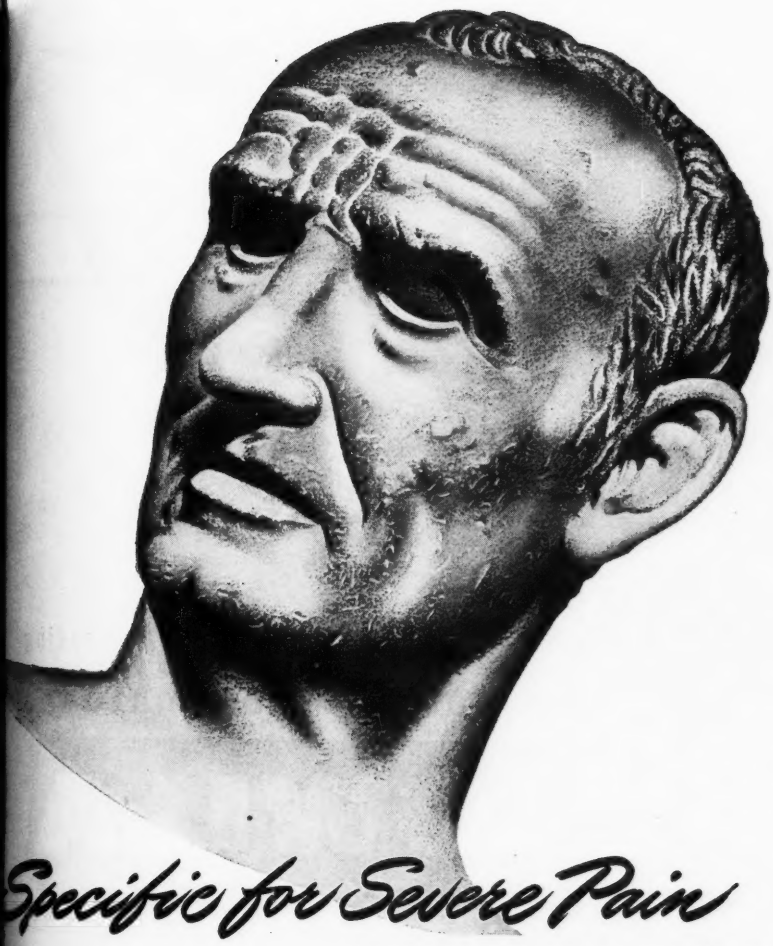

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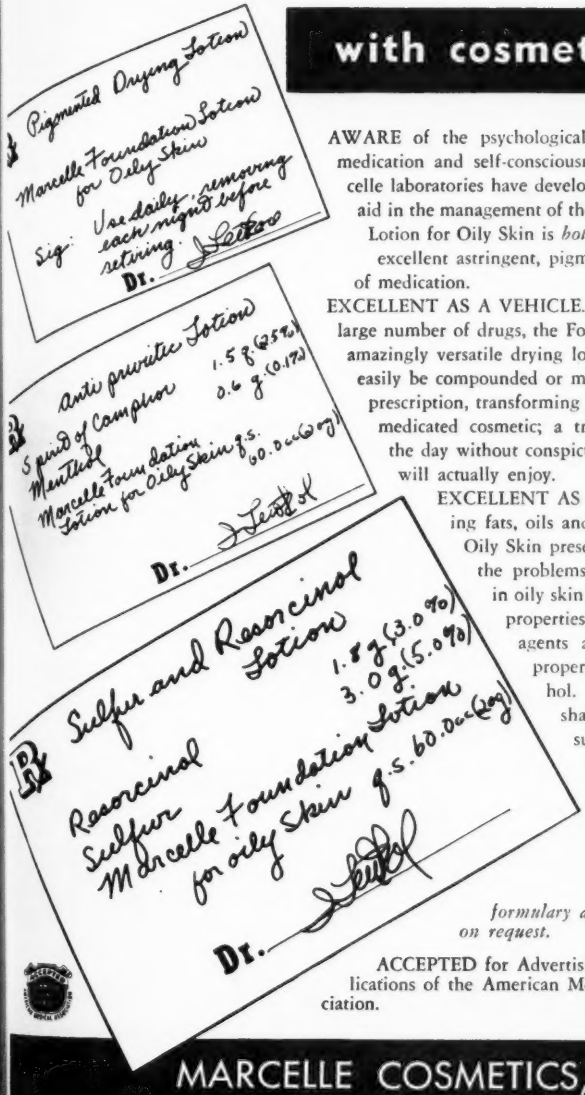

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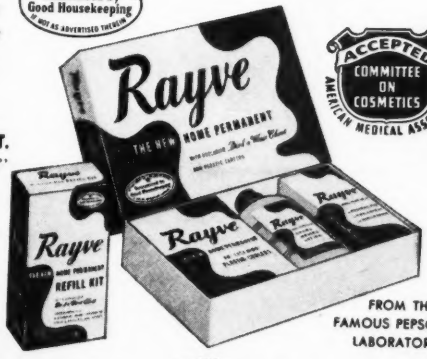

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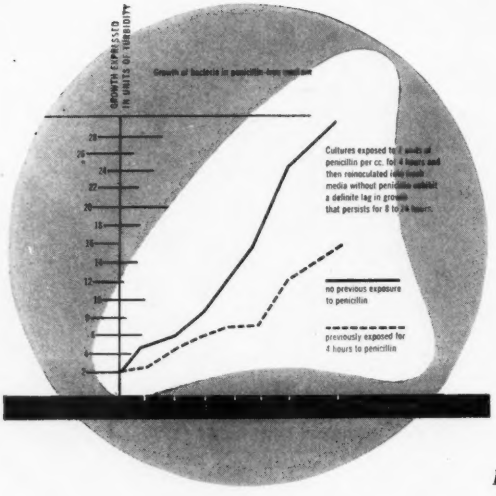
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VOLUME 60 DECEMBER 1949 NUMBER 6

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ROLE OF EMOTION IN DISORDERS OF THE SKIN

O. SPURGEON ENGLISH, M.D.
PHILADELPHIA

THAT emotion plays a role in disorders of the skin is a fact accepted by most, if not all, dermatologists. This fact has been well elaborated in the literature, both by those specializing in the field of dermatology and those in the field of psychiatry who have joined with dermatologists in thinking through some of the complicated disease conditions within that field of endeavor.

The psychosomatic approach to any disease makes a much greater demand for knowledge of causes, pathologic processes and procedures of treatment than any single newly discovered disease. This is because a whole new system of dynamic forces, emanating from the mind, which act as toxic agents, must be known and understood. Emotions can be helpful phenomena in the life of man, but they can also be harmful and make him sick. These emotions must be understood as causes of symptoms, with some ability on the part of the physician to assess and weigh them quantitatively. It must be known what kind of pathologic changes they will produce and also how therapeutic manipulation of these emotions will cause them to lose their capacity for producing end results of disease in the skin. Before passing to the role of emotions generally and what they can do, let us see what the commoner emotions are which are prone to be causes. The most commonly encountered emotions are the following:

1. Need for love (approval, appreciation, recognition)
2. Anxiety (fear and worry)
3. Hostility (anger, hate, aggression)
4. Inferiority feelings
5. Ambivalence
6. Guilt
7. Ambition (competition)
8. Envy

NEED FOR LOVE

The need of love is one of humanity's greatest hungers, and many other emotions are dependent on it. From the cradle to the grave human beings struggle under it. Some of them have a great hunger for love

From the Department of Psychiatry, Temple University School of Medicine.

but either have no recognition of what they need or have no technics for obtaining it. Lack of it brings such untoward emotions as frustration, hurt pride, envy and jealousy. Others have perverted and symbolic ways of obtaining it through excessive use of alcohol, food, sex or drugs. Others know they need affection, attention and rewards, and they take the conventional kind of actions and responsibilities which bring them the love they need and its derivatives of approval, appreciation and recognition. Thus, to be assured of love is a most important emotional need and sufficient gratification of this need is of the greatest importance to health, both physical and mental.

There has been a general reluctance to accept the proposition that love is an actual necessity for health and happiness. As a health factor it has not had the same scientific standing as the chemical symbol for iron, for example, but the more one studies man and his various illnesses, the clearer it becomes that he can live neither a healthy nor a wholesome life without it.

ANXIETY

Anxiety is one of the earliest emotions aroused and one of the most basic in the whole life of man. Fear is its conscious representative, but the origins of anxiety itself are largely unconscious. We have to use the word "fear" in order to describe the origins, since anxiety arises predominantly out of two early life situations, namely, fear of physical harm, pain or injury and fear of the loss of love. When the child is threatened with the loss of his mother's protective presence, he sustains uncomfortable sensations, in both the mind and the body, and in describing his reaction to this situation we say he fears a loss of love (meaning the loss of a friendly presence). Also, as the child comes to suffer pain from falls, bumps, rejection or punishment, he dreads its repetition. Its possibilities of repetition fill him with the same uncomfortable sensation, and we call this sensation anxiety. Anxiety has two components, one of them being psychic and distressing to the mind in a varying degree. The other, the somatic component, arises from the fact that once the distress of the psyche has reached the threshold of action the emotional energy overflows by way of the autonomic nervous system to any or all parts of the body. People who in the ordinary course of life suffer from too much anxiety are people who in early life lacked a consistent supply of a reassuring, friendly presence or who were subjected to too many real pain-inflicting experiences or threats of them. This allows a dread or a worry pattern to construct itself, and as life becomes more complex the things which threaten deprivation of love and security or real physical distress increase in number.

Not only does the emotion of anxiety impair one's ability to enjoy life, but it has more far-reaching effects. If one's energy is used up in

concentrating on the feared thing, in the hope that by this wariness it can be averted, too little energy is left for happy living. If this phenomenon goes on long enough and severely enough, the body can no longer function properly and symptoms of illness have arrived. These come through the mechanism I have already described, by which emotional energy is converted into somatic disturbances through the autonomic nervous system. We then have psychosomatic symptoms.

HOSTILITY

Like the need of love, hostility is also ubiquitous. Without spending too much time on the philosophic theories of the origin of hostility, let us say simply that when the human organism fails to find conditions which keep him in a constant state of well-being, he experiences an unpleasant emotion accompanied by ideas of retaliation and the use of force to gain his ends, even of destruction of the person or thing which makes him uncomfortable or which thwarts him. This we call hostility. It is impossible to conceive of any one in our society without any hostility whatever, but certainly small amounts can be present without too many untoward results. This is true, especially if the modicum is properly blended with the rest of the personality to produce wit or zestful, spicy conversation or if it results in a wholesome competition. "Get up and git," or "I'll show them," is an aggressive philosophy, which contributes to progress individually and generally. But, unfortunately for society, a great deal of hostility does not take this form. It does not even find vent in occasional outbursts of anger, righteous or otherwise. It has to be held in check, because the child or the adolescent does not feel that his environment will tolerate any show of hostility. He fears rejection or punishment; so his holding in results in repression—a "burying alive" process of which the mind is capable. The buried or repressed hostile activity builds up, and the more it builds up, the more labor another part of the mind has to exert to keep it from breaking out. While this battle is going on there is much less energy available for friendly relations in other directions. In fact, the person having this internal struggle becomes guilty about his state and not only feels no impulse toward loving but thinks that he does not deserve to be loved. The results of this continual psychopathologic engrossment may be various things, one example being the obsessive thought that he will harm somebody. However, we are more interested in where the hostile activity that builds up within expresses itself somatically. We discover that it goes in several directions to produce disturbances of the upper and lower gastrointestinal tract and enuresis, headache, ticlike movement, probably epilepsy and disorders of the skin, and plays a role in vascular hypertension and other disorders. Actually the effects of these

forces of which people individually appear so unconscious have long since been noted by them in general in that they observed that situations gave them "headaches" or "made them sick," or they "itched for a fight" and "got their blood pressure up" over something frustrating. Since people are not proud about being irritable, disagreeable, unpleasantly aggressive, arrogant, demanding, tyrannical or domineering, they remain remarkably unconscious of the hostile motivation. But there are tremendous quantities of hostility latent in the human race, producing not only serious social problems but many individual symptoms of illness as well.

INFERIORITY FEELINGS

To many it might appear that feelings of inferiority could exist as a social phenomenon but not one with any medical implications. But feelings of inferiority are closely linked up with the basic emotions of love and hate. Obviously, the person having feelings of inferiority has not had enough acceptance and appreciation. There are few people who ever feel quite satisfied with themselves, but in some persons the feeling of worthlessness can be so deep as to destroy the capacity of the psychic apparatus for maintaining physiologic equilibrium. Once a feeling of inferiority is deeply set, it is not easy to restore the self-esteem to a proper balance.

AMBIVALENCE

"Ambivalence" is a term given to the condition of directing both love and hate toward a person or the world in general. These impulses may be present practically continuously and show themselves rather subtly, or they may show themselves more superficially and plainly by a capricious change of mood and attitude, in which those associated with them are loved one hour or one day and hated and condemned the next. We see a man, for instance, who protests how much he loves his wife but never listens to her requests, her suggestions, never tries to further her plans and never helps her in any tangible way. We see a woman who protests that she loves her child but who never allows the child to do anything he enjoys and never lets him decide anything for himself. This mixture of emotions which has the descriptive term of ambivalence, which portrays it as one emotion, can, like guilt, block off the benefits of straightforward, friendly, satisfying and wholesome human relations.

GUILT

Guilt is an emotional distress resulting from criticisms voiced by the conscience, the conscience being a well preserved memory pattern of what the parents or substitute mentors said was right and proper. The person undergoing guilt has reason to feel that he has not been or is not

living up to expectations. This emotion can kill the joy of living and may cause the patient to adopt a life of excessive self-sacrifice, which in the course of time produces physiologic disturbances. As an emotion it acts to block the reception of impulses emanating from others of love and approval. Starvation of the love needs can occur from guilt and the pathophysiologic results in the way described earlier.

AMBITION (ENVY, COMPETITION)

The ambitious person does not necessarily have pathologic emotions, but he certainly runs the risk of being infected with envy or excessive competitiveness. These emotions tend to produce tension, and the professional, financial and social success of many people has been paid for at a high price, i. e., the price of tension, which is prone to express itself through the nervous system on many parts of the body. The aggressive component inherent in these emotions plays a large role in such conditions as cardiovascular disease, migraine and vascular hypertension, and to some degree in many others. A successful person can have achieved his goal by great ability and a friendly, easy-going manner, but he is in the minority. Ambition with its attitude of competition, sometimes accompanied by envy, is all too often carried along as unnecessary equipment on the road to successful service.

With this section I conclude these brief pictures of the more common and important emotions which are going to affect the skin, if emotion is playing any role whatever.

HOW THE EMOTIONS AFFECT THE SKIN

Since emotions will find expression in any or all parts of the body, it is inevitable that so important an organ as the skin should be affected. Probably the greatest number of ideas relating to man's actions and feelings are spoken of in reference to the heart. The stomach and bowel run the heart a close second, but the skin will not be too far down the list. When, for instance, a man does anything free of blameworthy motives, we allude to him as acting "with a whole skin." When we escape injury, we "save our skin." Cheating or taking advantage of the other fellow is to "skin" him. To be oversensitive is to be "thin-skinned" and to be relatively impervious to the attitudes of others is to be "thick-skinned." The expression "you are a scab" or "you give me the itch" are well known and indicate the response of the skin to hostility. "I blush for you" means "I identify myself with you and disapprove of you." Such expressions indicate that the skin is a potent carrier of emotions and attitudes. Just as the stomach may act in a perverted way when the personality is unable to cope with a problem, so the skin may do the same. Our first impression was that emotion was slower to appear in the skin than in the heart or the gastrointestinal tract, but

in blushing and sweating we have very prompt phenomena which sometimes are distressing to a repressive patient. Also, urticaria and allergic phenomena have a high incidence and can make their appearance quickly. Hence, so far as these two phenomena often have a large emotional component, we cannot say that the skin differs from other organs because it does not respond promptly. Dermatology and psychiatry share the phenomenon of having a few acute conditions and many which are slow to improve. Now the skin undoubtedly is like other organs in that it may symbolically represent an inner attitude. In other words, the skin may give one away. The well known phenomenon of blushing embarrassment indicates "I believe you know what I'm thinking and I'm afraid and ashamed." The blush symbolizes inner excitement with fear of exposure being prominent; the exhibitionistic wish, however, triumphs over fear and shame, which would try to keep these emotions hidden.

But the skin, in addition to "burning" with shame, may weep out of sadness. So just as there may be an idea quickly speaking its message by way of the skin, so long-continued tensions make themselves known through activity of the autonomic nervous system. In adolescence, for instance, there are many conflicting feelings of being good and bad, of being attractive and ugly, of being clean and dirty, of being intelligent and stupid, all at the same time. The restless longing for attention makes them latently strongly exhibitionistic. It is a paradox that at the time they wish to be most presentable, attractive, lovable, beautiful, they are affected with acne or some other disturbance of the skin. But if we grant that emotions affect the function of the skin at all, then it becomes plausible that they must show their worst side through disease of the skin. Both children and adults may expose their worst behavior in a bid for attention. The skin may take over this action. What we must keep trying to do if we are to be successful in understanding the language of psychosomatic disease is to remember that the skin can act a certain way or act out a problem, just as the whole person may act through voluntary muscle activity.

Since most physicians pride themselves on being practical men, these ideas may sound impractical and fantastic to many. However, the practice of psychosomatic medicine requires that the physician use other aspects of his education than physics and chemistry. It requires that he (1) utilize what he knows or can learn from the poet, the story writer, the artist and the philosopher and (2) bring this knowledge to bear on his patient's approach to the symptoms in question. This approach seems less reliable and is certainly less tangible than remedies which can be taken internally, intravenously, intradermally, intramuscularly or by unguentation. It takes a great deal of understanding and self-confidence to ask the patient some such question as the fol-

lowing: "What is your skin trying to say for you?" or "What is there of your inner self which is revealing itself through your skin?" or "What is your skin expressing?" or "What do you suppose your skin is acting out?" The physician has to be very comfortable with the idea that personal feelings and experiences will use body organs for expression. Only when the physician believes that such things can happen and is personally comfortable in his own thinking about these concepts, can be bring the patient to make his contribution in treatment cooperation.

NEUROPHYSIOLOGIC CONNECTIONS BETWEEN EMOTION AND SKIN DISEASE

Now it is unlikely that there is any symbolic expression of ideas or attitudes anywhere in the body which does not utilize nerve pathways, vascular changes, chemical alterations and structural tissue changes as the medium for transmitting the message. In some cases the psychic stimulus acts over a short period and then wanes in strength or is neutralized by a counterphenomenon, as, for instance, the homesick man or woman who is able to return home. In other cases the stimulus acts continuously over a prolonged period and is neutralized with difficulty or not at all.

Having enumerated the emotions and in a general way indicated their mode of action, let us turn to the emotional states in which we encounter a linking of emotions and skin problems.

NEUROTIC EXCORIATIONS

As knowledge of human emotional needs and how they are met increases, the line of division between what is neurotic and what is psychotic becomes less important. There is a pattern of wholesome development of personality and adaptation, and if this pattern is not approximated the many devious by-paths of attempted solution are almost legion. In neurotic excoriations, for instance, the patient may already be suffering from delusions of parasitosis before the behavior which produces the excoriations begins. Even if delusions are not present, there is a strong need to disfigure, to suffer pain, to call attention to the self, to seek attention in a perverse way because the more healthy, wholesome way was never discovered and firmly established early in life. The wholesome pattern of training tends toward beauty, culture and the esthetic satisfaction of others. This brings rewards of ego satisfaction, but if they are unknown to the patient and if he knows and seeks pity, shocked surprise, anxious concern or even revulsion and condemnation, he nevertheless accepts this reaction in others rather than being a complete nonentity in their eyes. Most people cannot stand being ignored, and self-mutilation producing an unsightly skin,

like a social act of bravado, is preferable in certain egos to the emptiness of oblivion. If delusions are present as the basis of self-inflicted excoriations which follow, we should, instead of speaking of the role of emotion in skin disorders, speak of the invasion of morbid ideas when healthy emotions and ideas are absent. Happy people, i.e., people with a sense of well-being, tend to seek out others and join in mutually pleasurable behavior. It is when this sense of well-being is absent that an individual falls back on himself, feels himself, "tunes in on himself" to use the expression of one patient and falls prey to ideas of something morbid or unhealthy in his skin or some other part of the body.

DERMATITIS FACTITIA

In dermatitis factitia we see the patient secretive about his goal. He wants attention, but one must be a detective and guess he wants it. He even tries out one's diagnostic acumen like the children in New York who recently ran away from home and hid in a cemetery and when found by detectives said "we just wanted to see if you were any good at your business." The patient with dermatitis factitia seems to enjoy challenging the physician to make a diagnosis for the pleasure of being enigmatic (to see if the physician knows the business of understanding people) before the real question of the motive of self-mutilation ever becomes an issue.

EMOTIONS AND SKIN SENSITIVITY

Eczema.—There is increasing evidence that maternal rejection plays a large role in psychosomatic conditions, just as in neuroses and psychoses.[1] In fact, we do not mean to distinguish between these three divisions of emotional conditions too sharply, for clinically they merge as different manifestations of the same basic emotional and ideational difficulties—each merely having its own pattern and intensity. This maternal rejection with its absence of affection to meet the minimum needs of love seems to leave the skin organ in a state of sensitive irritability or easy vulnerability to internal emotional conflict or external pressures or deprivations, just as the same conditions disturb heart action and normal function of the gastrointestinal tract or even prevent restful sleep. Without the buffering or protective effect which a restful, secure relationship with the mother produces, we have a skin which might be subject to eczema or urticarial symptoms without there being an immunologic allergy in the patient.[1] Miller and Baruch make a distinction between patients in whom asthma, hay fever, eczema and urticaria may have immunologic allergy as their source and those patients in whom

1. Miller, H., and Baruch, D. D.: Psychosomatic Studies of Children with Allergic Manifestations, Psychosom. Med. **10**:275, 1948.

the symptoms may have nothing to do with immunologic allergy. They feel that psychologic elements do not produce immunologic allergy but that in many patients similar symptoms may certainly not be the result of immunologic allergy. Such patients who are not allergic in the immunologic sense would, we assume, have this sensitivity of skin in the same way that some patients have a most labile heart action, others have a sensitive stomach and others are sensitive sleepers. We are especially indebted to Miller and Baruch for including in their study a control group of children. Further studies of this kind with controls are much needed in this field of our mutual interest. This would mean that when the child or the adult was under stress or when the relationship of the child and the mother or her substitute was endangered, the skin would be vulnerable to symptomatic reactions.[2] Certainly, we observe frequently enough inflammation, itching and weeping of the skin in eczema when the patient is going through some environmental stress, which places him in a position of responsibility where he feels alone and unprotected. Then he has a longing expressed in the skin by itching which calls for the soothing hand. (The hand which scratches is the soothing hand, temporarily at least.) He cannot weep, but his skin weeps for him. Eczema patients are usually depressed and long for love, but they can't use love when they get it. It may even tend to aggravate them. They long to scratch instead, but they are only the more aggravated by scratching. After all, love has been defined as "an itch one can't scratch." So far as itching may represent a desire for love, certainly scratching is an unsuccessful attempt to obtain it.

PRURITUS ANI AND VULVAE

A later period of emotional fixation would be when in the course of psychosexual development, the potential pleasure for sexual excitement and orgasm never fully reached the genital region. It remained in the anal region and scratching served the role of a sexual excitement, instead of the mature genital pleasure. Pruritus ani and vulvae is not always psychologic in origin, but a psychologic factor doubtless plays a contributing role in many cases. That this is true does not preclude some capacity for normal sexual function in the individual with the condition. The case is not, in other words, one of a normal or a perverse way of sexual gratification, but it can include a little of both, and this should be investigated as a possibility in each case of pruritus. More than one patient has said such things as the following about pruritus, "It's like being excited sexually, only one is never able to achieve an orgasm. It's only excitement and tension and no let down."

2. Saul, L. J.: Relations to Mother as Seen in Cases of Allergy, Nerv. Child. **5**:332, 1946.

CANCER PHOBIA AND SYPHILOPHOBIA

These conditions are not actually cutaneous conditions but psychiatric conditions and severe ones at that, but since their nature is such as to bring them to the attention of dermatologists, some discussion of them seems pertinent in a paper of this kind. Just as maternal rejection may sensitize the skin to minor or major departures from peaceful and quiet functioning, so the same state of affairs prevents an emotional buffering from morbidness of mood. When a person feels unloved and does not have the interest of those who have power and wish to protect with that power, ideas spring up in the mind of destructive forces invading the organism. In childhood it is the bogeyman or wild animals, while in adulthood it is the undue suspicion of burglars and violence or of invasive cancer or syphilis. Syphilis or cancer is merely the later life formulation of the bogeyman or the wild animals. In either case, pain, destruction of tissues and eventual annihilation are felt to be the end result. In phobias there is a great distrust born of real events in early life which would normally evoke distrust. There are guilt and feelings of unworthiness. There is great difficulty, amounting to almost complete inability in some cases, to trust and believe in the one who would help them. Out of a long-carried-over feeling of guilt they think, "I should be the victim of a destructive disease. I deserve no better fate. Something dreadful must happen to me to even the score. I am being, or I will be, punished for my wicked thoughts and acts." In therapy the task is to have the patient feel a warmth toward others and a joy in doing for others, so that he begins to reestablish a feeling of worthiness. This must provide the ground work for interpretation of the symptoms. No belief in a worried person is senseless. Every belief has a cause in some earlier experience. There is not necessarily one thought or fantasy which, if discovered, enables the whole neurosis suddenly to disappear. This happens only rarely. In these cases most of the results depend on slowly enriching the empty emotional life, which has left room for morbid ideas, and coming to an understanding of why a painful thought like that of having cancer or syphilis can serve any purpose. It serves to atone for some childhood misdeed or "bad thought." Only when this is understood and the patient can forgive himself, can he understand how to have a life free of the burden of constant preoccupation with such distressing thoughts. Some of these patients improve and get well with a few psychotherapeutic interviews—three to six in number. Most of them, however, if their disease is of more than six months' duration, need longer, more intensive psychiatric treatment and should be referred to a psychiatrist for treatment, if feasible. These cases can be among the most difficult in the field of psychiatry.

TRICHOTILLOMANIA

Trichotillomania is likewise not really a condition of the skin but a piece of neurotic behavior. What one sees in the hairy regions are the end results of a compulsion to extract the hairs forcibly with the fingers or other means. This unusual behavior starts as a perverse means of gratification, of producing, in other words, pleasurable pain. Those whose behavior is of this sort are in the class of those who bite and pick the nails until the nails are painful and bleeding, crack their finger joints, stimulate the gums with toothpicks or other instruments until the gums bleed or who squeeze and press comedos until they have produced macerated areas and bruises. Tugging at ear lobes, biting the lips or pinching the skin are other manifestations of efforts to produce a pleasure-pain effect on the body, because more natural and wholesome ways of relieving tension either have not been permitted or have not been taught early in life. They play with themselves, stimulate themselves and usually disfigure themselves, mainly because the activity gratifies. This fact is hard for average people to understand, but it is no more enigmatic than the fact that people consistently eat or drink more than is good for them. In either case they (1) indulge themselves in something they enjoy, (2) they relieve tension at the moment, (3) they put themselves at a disadvantage in the eyes of others through it but (4) there is an insistent urge to repeat it for the momentary pleasure it produces. From this description its sexual component can be seen. To cure it, the physician has (1) to help the patient to endure the tension on cessation of the habit and (2) to get the energies channeled into more wholesome and useful directions.

HYPERHIDROSIS

Excessive sweating of the hands, axillas and feet is fairly common. It probably could not be settled at the present time whether there is any inherent pathologic condition of the sweating mechanism or whether this manifestation is entirely emotionally conditioned. There is considerable evidence for the latter. There seems every reason to believe that the sweat gland mechanism might have as wide variation in function as the glands in the mucous membrane systems of the stomach, the intestine and the respiratory and genitourinary tracts. One must grant that there is wide variation in the outpouring of secretion in these areas, and the skin response could be just as labile. We see analogies in sphincter control, where in states of fear people cannot "hold their water," and it drips from the hands, axillas and feet and often other parts of the body as well. Whether there is any wisdom of the body in this mechanism; that is, whether there are any conditions under which

this would be advantageous is unknown to us. MacKenna [3] suggested that "a patient suffering from pompholyx may be indicating by a psychological mechanism that he cannot use his hands and feet." However, in psychiatry, when hyperhidrosis is an accidental symptom of an otherwise more inclusive neurotic pattern, we frequently find that in the course of gaining the security which cures the other symptoms, hyperhidrosis ceases. In this connection, Wittkower,[4] in a study of 50 patients with pompholyx, found them to be unusually narcissistic, and four-fifths of them had shown other outstanding neurotic symptoms prior to or at the time of onset of the condition. He found them vain and affected in their manners and speech. They were ambitious but afraid of failure. They were afraid of getting hurt physically and emotionally. They were afraid of exposing their own feelings to others. If severe hyperhidrosis exists alone as the main complaint and is unresponsive to local treatment, psychotherapy should surely be utilized. As in blushing or more particularly the fear of blushing (erythrophobia), the patient may be reluctant to spend the time necessary to get at the emotional basis of a symptom which appears simple in nature but actually has a great deal of psychopathologic material underlying it. Should the patient decide to undergo intensive psychotherapy for a symptom like pompholyx or erythrophobia, there is no question but that his personality would derive great benefit as a result of the changes he would have to make to get rid of his symptom. The patients rarely realize that they need to get any help with their personality, generally feeling adequate in this area. However, in making this need clear to the patient we may help him to decide that the treatment for the removal of just one symptom, which at first glance looks tedious and expensive, should be undertaken. Just one such symptom may render a patient ineffectual and miserable; so the fact that the treatment offers two benefits may be the means of getting him to accept and participate in the therapeutic program.

URTICARIA

Urticaria is one of the conditions of the skin for which an emotional factor has been noted and accepted for some time. As yet it becomes difficult to specify the emotional trends which, among other factors, enter into this condition. Stokes, Kulchar and Pillsbury [5] concluded

3. MacKenna, A. M. B.: Psychosomatic Factors in Cutaneous Disease, Lancet **2**:679, 1944.

4. Wittkower, E.: Psychological Aspects of Skin Disease, Bull. Menninger Clin. **11**:148, 1947.

5. Stokes, J. H.; Kulchar, G. V., and Pillsbury, D. M.: Effect on the Skin of Emotional and Nervous States: Etiological Background of the Urticaria with Special Reference to the Psychoneurogenous Factor, Arch. Dermat. & Syph. **31**:470 (April) 1935.

from a study of 100 cases that the patients were of the driving, high tension, competitive type, keyed to high pitch and perpetually intent on a destination to be achieved at no matter what expense. Saul [6] seemed to find that longings for love frustrated through fears of sex in persons with a highly erotized skin were responsible for attacks. Both observations have the common denominator of intense longings for achievement of gratification. If we keep in mind that there are many hard-driving people who long for things without getting urticaria, but that it is this type of personality which furnishes the final factor that sets the symptom complex into motion, and that this factor can be treated and tension in this direction lowered, we have done the most fruitful thing possible toward relief of the symptoms.

SEBORRHEIC DERMATITIS

Ingram [7] in 1939 called attention to the emotional instability of those suffering from seborrheic dermatitis by stating that it could vary from the sullen, sulky indolence of patients who present some of the heavier forms of acne to the high-strung overactivity of the patient with rosacea. "States of anxiety, stress, and strain . . . are conditions which the seborrhoeic will not readily tolerate." Ingram studied 100 seborrheic patients and found them hard to study, as they were not easy talkers and were afraid of persons in authority. They were shy and solitary and had trouble in social contacts. When with people they felt lost, exposed, uneasy and tongue-tied. They could not stand hostility toward themselves. They were compulsive workers and perfectionistic. They were sensitive and felt inadequate. As might be expected from such traits, they were unaggressive and overcompliant. No follow-up treatment is reported, but it could be assumed that any cutaneous symptom present with such marked disturbances of personality should improve under effective therapy.

The same author studied patients with psoriasis and found no specific type of personality, but concluded that the proportion who were maladjusted was far in excess of the proportion of such persons in the general population.

As the role of emotions in cutaneous conditions becomes more widely accepted, we shall have more psychotherapeutic efforts and hence more reports of results of treatment. This will be good for the morale of both dermatologists and patients alike. As things stand, the patient still has too little faith in what can be done for his condition by a psychotherapeutic approach. It is really hard for him to look at his emotions,

6. Saul, S. J., and Bernstein, C.: The Emotional Settings of Some Attacks of Urticaria, Psychosom. Med. **3**:349, 1941.

7. Ingram, J. T.: The Seborrhoeic Diathesis, Brit. M. J. **2**:5, 1939.

since he is too busy looking at his skin and sees little reason why he should shift his attention to his emotions, which he considers as secondary to the state of his skin. But as word gets around that one's disposition can be expressing itself in the skin, the physician can obtain more cooperation in the psychotherapeutic treatment.

The emotional factor is a variable one, and no one is trying to assert that skin diseases are due to emotions alone. But when emotion is a factor, it is one that must be dealt with; if well dealt with, it may be the factor which will give the most practical results in comfort and hence in satisfaction to both patient and physician. Dermatologists and psychiatrists must stimulate and help each other to carry on more therapy of the emotional forces which are using the skin for their expression. Psychotherapy is not a simple, easily mastered tool, and the patient is not always a willing subject for therapy. But, if we try to ascertain what the role of emotions can be in conditions of the skin, we will cure the condition in occasional subjects, improve it in some, make it easier to live with in others and find the specialty of dermatology altogether more interesting through the pleasure which comes from taking a greater interest in human emotions in our daily work as physicians.

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OLD DERMATOLOGIC DRUGS WHICH SHOULD BE RETAINED

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WE ARE living in the "Golden Age" of dermatologic therapy, or so it would seem. During the decade since sulfanilamide was first introduced into therapy a procession of new potent drugs has been added to our weapons against disease. With the sulfonamide compounds in all their many variations; with penicillin, streptomycin, tyrothricin, bacitracin, aureomycin and other antibiotics yet to be named; with the vitamins in all their fractionations; with radioactive isotopes, and with the host of antihistaminic drugs it would seem that the therapeutic progress in these few years is unmatched in any period in history. Only time and the merciless inroads of experience will tell whether these contributions will live through the ages as permanent achievements, as have the artistic wonders of the "Golden Age of Pericles."

It is unfortunate that all these drugs have appeared on the scene as "wonder drugs." Such an introductory appraisal is not conducive to the competitive emergence of a drug of permanent value. Each drug was wishfully accepted as a miracle and at once was utilized in every conceivable disease. It was only after mishaps, complications and failures that the faith in miracles was shaken and was replaced by the cold, analytic scrutiny that should be the challenge of all new drugs. There is much evidence already on record that not all the advances made by the use of these new drugs are permanent steps forward. In the problem of infectious diseases there is a continuous adaptation of the micro-organisms, which in time prevents their complete annihilation. Many of these drugs produce sensitizations or other toxic reactions which make their continued use hazardous. It is true that all the new drugs are being constantly developed and improved. While improvement is always a sign of progress, it is nevertheless also an indication that there are deficiencies that need correction.

In times like these, when to the casual observer we dermatologists seem to be moving from success to success, it is well for us to look backward, to appraise the past and to hold fast to what is good. In

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former generations, when nothing was known of the causes of most diseases of the skin, an attempt was made to treat the conditions symptomatically. Because of the surface location of cutaneous lesions it was possible for physicians to experiment widely with all possible local medications and, by trial and error over the years, to develop topical therapy that was effective. Much of accepted dermatologic therapy is based on this tediously developed but long-established foundation.

In modern times, when very little more is known about the causes of most diseases of the skin, the emphasis has been placed on etiology. The goal set has been to achieve specific treatment pointed to correcting definite etiologic factors. This orientation may in time result in a brilliant solution of many of the baffling dermatologic problems. There is the drawback, however, that we may for too long be withholding relief of symptoms and even possible cure by so-called shotgun therapy while we spend precious time in taking careful aim with a precision rifle—a weapon that in many instances may not even be loaded.

It has been my impression that with the frequent addition of well publicized new drugs to the dermatologic armamentarium, many old ones have undeservedly been pushed into the background of daily thinking. Dermatologists now being trained are often unfamiliar with old drugs that still have merit, and they tend to rely more and more on therapeutic weapons that may prove to be ephemeral. With the purpose of emphasizing those drugs that have proved to be effective over the years in the work of successful practitioners, I addressed a letter to two hundred dermatologists of experience, asking them to list drugs of long-established usage that they still prescribe. Replies were received from nearly all. Most of those questioned were generous in listing the drugs they use most often in their work. One dermatologist referred me to the "Pharmacopoeia of the United States" (U. S. P.) and the "National Formulary" (N. F.) for drugs that still have merit. I heartily agree that one may practice in our specialty very competently while limiting himself to drugs listed in those two authoritative books. It is, nevertheless, true that in our Academy there are many who do not have in their offices, available for easy reference, the latest edition of either work, nor even that excellent small book, the "Epitome of the Pharmacopoeia of the United States and the National Formulary." I am glad to pass on the suggestion, however, and to recommend the "Epitome," always in the latest edition, as a book that should be accessible on every dermatologist's desk. A careful study of its contents is rewarding in calling to attention many official preparations, easily compounded and readily obtained, that are useful in the treatment of many dermatoses. Most of these preparations have the added virtue of being relatively inexpensive.

Nearly every dermatologist of experience has his pet remedy for each dermatosis, a drug tested by time and countless usages and at times inherited from his preceptor. While the use of some of these remedies may represent wishful thinking and unwarranted enthusiasm, often it represents the product of careful observation of cause and effect by good clinical observers, whose experience should not be lightly brushed aside. In the practical management of a dermatologic patient it is helpful for us to keep all these remedies in mind. Far too often we dermatologists exhaust all methods of treatment which are indicated on rational grounds and in the end come up against the stark fact that our patient has not been helped at all. It is helpful at such a time to be reminded of drugs that empirically and often without logical justification may succeed in giving the patient at least symptomatic relief.

DRUGS FOR INTERNAL USE

Of the drugs used internally arsenic compounds probably have the oldest history and the most varied career from generation to generation. Centuries ago they were used as a remedy for asthma. For many years they fell into disuse medically because of frequent employment as a poison. In the seventeenth and eighteenth centuries they were used medicinally only by quacks for treating fever and ague. One of these quack preparations was so successful that a Dr. Fowler had it analyzed and published the prescription in London in 1786. The use of Fowler's solution (potassium arsenite solution U. S. P.), as it has since been known, helped to raise arsenic compounds again to a status of medicinal respectability. One need not discuss at length the importance attained by arsenic compounds with the introduction of arsphenamine ("salvarsan") by Ehrlich for the treatment of syphilis. This importance was enhanced by the later development of neoarsphenamine (neosalvarsan®) and the more recent oxophenarsine hydrochloride (mapharsen®). At one time or another arsenic compounds were employed in the treatment of nearly all dermatoses. They undoubtedly have some beneficial effect in many diseases. Their general use was soon limited, however, by the recognition of their occasional harmful effects, such as keratoses, epitheliomas, arsenical dermatitis and visceral complications. In the treatment of syphilis, however, arsenic compounds long remained unchallenged. With the advent of penicillin, it seems at present as if arsenical drugs will be replaced completely even in the treatment of that disease. Only time will tell whether treatment with penicillin, effective as it is in quickly rendering a syphilitic patient noninfective, will need to be followed by chemotherapy in order for one to obtain the highest possible percentage of long range cures or remissions.

Arsenic compounds are still being used frequently in diseases other than syphilis. Neoarsphenamine and oxophenarsine hydrochloride are

used in the treatment of sarcoid, tuberculosis indurativa, papulonecrotic tuberculid and lupus erythematosus, often producing considerable improvement. Injections of solutions of ferric cacodylate or sodium arsenate have been employed in the treatment of pemphigus. Acetarsone tablets taken by mouth have at times been effective in the treatment of pemphigus and lichen planus. Asiatic pills (composed of arsenic trioxide and black pepper) and potassium arsenite solution, however, have been the most popular forms of administering arsenic by mouth, and they have been used especially in the treatment of lichen planus, dermatitis herpetiformis, neurodermatitis, urticaria papulosa, pompholyx, parapsoriasis and vitiligo. In the treatment of psoriasis the use of arsenical drugs should be discouraged, for they need to be administered for too long a time to be devoid of danger. It must be remembered, however, that in some persons the use of arsenic compounds for even a short period may be followed by serious sequelae.

Iodine in the form of a solution of potassium iodide given by mouth is still an important adjuvant treatment of late syphilis. It has also been recommended as an adjuvant to streptomycin in the treatment of tuberculosis. It is an excellent remedy in cases of blastomycosis, sporotrichosis, actinomycosis, kerion celsi and other deep fungous infections. At times it is effective in the treatment of arthropathic psoriasis or resistant psoriasis, a use which is seldom remembered at present. In the form of strong iodine solution U. S. P. or iodoform pills it is helpful at times in treatment of cases of lupus erythematosus. Intravenous injections of a solution of sodium iodide often relieve the pain of herpes zoster.

Mercurial preparations are seldom used at present in the treatment of syphilis. As a therapeutic test in late syphilis, however, the oral administration of mercury and chalk tablets is very useful. The oral administration of mercury with chalk or yellow mercurous iodide is often effective in the treatment of juvenile warts and lichen planus, while mild mercurous chloride is an excellent cathartic in the treatment of urticaria and toxic erythema.

Bismuth compounds given by intramuscular injection have long been used in the treatment of syphilis. While at present displaced by penicillin, they are still being employed in the supplementary chemotherapy of that disease. In lupus erythematosus and lichen planus they are often effective, either alone or combined with arsenic as in bismuth arsphenamine sulfonate (bismarsen®).

Gold, in the form of intravenous injections of gold and sodium thiosulfate or other gold compounds, is still probably the most effective drug in the treatment of chronic discoid lupus erythematosus, although the percentage of failures with its use is increasing as the years go by.

Antimony, as antimony potassium tartrate or stibophen (fuadin®), seems to be outdated in the treatment of granuloma inguinale as a result of the introduction of streptomycin. Antimony potassium tartrate is still being used, however, in treatment of cases of mycosis fungoides and pemphigus foliaceus.

Intravenous injections of a 10 per cent solution of sodium thiosulfate are sometimes of benefit in the treatment of disseminated neurodermatitis. In the treatment of arsenical exfoliative dermatitis, however, that drug has largely been displaced by BAL (2,3-dimercaptopropanol).

Sodium salicylate given by mouth is often effective in treatment of cases of erythema multiforme and erythema nodosum, while phenyl salicylate is more often recommended for urticaria, and salicin for psoriasis. Acetylsalicylic acid, alone or in combination with codeine, forms one of the most effective drugs for the relief of pain in herpes zoster.

In the treatment of urticaria and other allergic dermatoses the antihistaminic drugs seem to have successfully eliminated all drugs used formerly. In the urticaria that results from penicillin, however, epinephrine is often necessary for quick relief and at times ephedrine is also helpful. In individual cases benefit may also be obtained after the employment of bile extracts, belladonna tincture, cascara sagrada, magnesia magma, mild mercurous chloride, phenobarbital or calcium gluconate injection.

Quinine has been used in the treatment of severe acne vulgaris and resistant cases of lupus erythematosus.

Foreign protein therapy, in the form of administration of bactalbumin (aolan®), a solution of peptones and proteoses derived from milk (proteolac®), milk, intravenous injections of typhoid vaccine or autohemotherapy, is often of great value for severe acne vulgaris, neurodermatitis, dermatitis venenata, psoriasis and dermatitis herpetiformis, although for the last-mentioned disorder sulfapyridine has proved to be the most effective drug in cases in which it can be employed.

Of the sedatives, phenobarbital is probably the most widely prescribed drug, but at times bromides, chloral hydrate or paraldehyde are more effective when prolonged sedation is desired.

Sodium chloride is indicated in the treatment of bromoderma and bromine acne. It is effective in the enteric-coated tablets, and it seldom needs to be given intravenously.

Vaccine therapy still has its supporters in treatment of recurrent furunculosis and pyodermas, and its employment is not completely outdated by the introduction of penicillin and the sulfonamide compounds. Smallpox vaccine is often beneficial in cases of recurrent herpes zoster.

Of the endocrine substances thyroid is probably used most often. It is especially indicated in the treatment of myxedema and hypothyroid loss of hair, but it is also prescribed for scleroderma, onychodystrophy and, at times, for acne. Posterior pituitary injection (pituitrin®), 0.25 cc. (obstetric dose) given subcutaneously daily, often relieves the pain in herpes zoster and hastens the involution of the disease.

Dilute hydrochloric acid is prescribed in cases of rosacea accompanied with achlorhydria.

Iron compounds given by mouth, hematinic tablets and injections of crude liver are often valuable in the treatment of severe acne vulgaris, lupus erythematosus and pemphigus, but in the last-mentioned disease a blood transfusion is probably the most rapid procedure for improving the hematologic status of the patient.

Preparations of the vitamin B complex, dried yeast and extract of rice polishings are used in the treatment of glossitis, perlèche, pellagra and rosacea, while vitamin A preparations are often beneficial in treatment of cases of keratosis follicularis, keratosis pilaris, ichthyosis and pityriasis rubra pilaris.

DRUGS FOR TOPICAL USE

It is in topical therapy that we dermatologists have our greatest heritage. It is in this field also that great skill in therapy can be acquired most rapidly by the intelligent interpretation of experience. The surface location of cutaneous lesions, the ease of applying many forms of medication and the ready observation of the effects of local applications make the skin an ideal experimental organ. A novice who studies minutely the clinical features of all cutaneous lesions and remembers well how they react to different drugs in various concentrations and in diverse vehicles soon has acquired the therapeutic wisdom of a veteran. The careful selection of a few effective drugs and their adaptation to the treatment of various diseases in all stages of activity is much more educational than an aimless but wishful flitting from one proprietary mixture to a later one.

In cases of generalized pruritus baths may be used to make a patient more comfortable. Cool colloid baths, prepared with starch, bran or oatmeal, are soothing to an inflamed skin. Tar baths are antipruritic and at times are also helpful in treatment of extensive psoriasis. Baths containing potassium permanganate are useful in the therapy of widespread pyodermas and of pemphigus.

Cold wet dressings are often the most soothing form of treatment in acute inflammations of the skin. Skim milk containing boric acid is nearly always well tolerated. A saturated aqueous solution of boric acid is effective and inexpensive, while aluminum acetate solution

(in a dilution of 1 to 16) and a combination of the two are extremely popular. In treatment of dermatitis due to plants, solutions of lead acetate (1 teaspoon to 1 quart), potassium permanganate (1 to 5,000) or strong tea generally bring relief. In treatment of pyoderma or acute tineal infection of the feet alibour water (water containing zinc and copper sulfate; 1 to 20) or potassium permanganate (1 to 5,000) are of great value. Solutions of silver nitrate (1 to 500 or 1 to 1,000) are efficacious in the therapy of acute oozing dermatitis and the weeping streptococcic dermatitis seen on the legs or behind the ears.

Of the drying lotions, calamine lotion and starch lotion, with or without the addition of phenol and menthol, are the most widely used. When the skin is to be kept from becoming too dry, calamine liniment or lime liniment may be used.

Most powders contain starch, boric acid and talc, at times with the addition of other medicaments for specific purposes, such as salicylic acid and thymol in treatment of tineal infections.

Soothing ointments include petrolatum, rose water ointment, hydrous wool fat, theobroma oil, lead oleate ointment, zinc oxide ointment and zinc oxide paste.

Effective antiperspirants are a 25 per cent solution of aluminum chloride, a 1 per cent solution of formaldehyde and, for the feet, a 1:4,000 solution of potassium permanganate.

Mercury is still one of the most efficacious drugs for topical use on the skin. Except for the few persons who are sensitive to the drug, ammoniated mercury in the form of a 3 per cent to 10 per cent ointment is probably the most suitable medication for impetigo, ecthyma and other pustular infections of the skin. Five per cent ammoniated mercury ointment is often rapidly effective in treatment of infected eczema of the auditory canal, and it is also well tolerated in treatment of impetiginized atopic dermatitis. Its use is often beneficial in cases of psoriasis and seborrheic dermatitis. One per cent ointment of yellow mercuric oxide is useful in treatment of seborrheic dermatitis of the eyelids, and in therapy of pediculosis of the eyelids it is unexcelled. A 1 to 5,000 aqueous solution of mercuric oxycyanide is also an effective and clean treatment for seborrheic dermatitis of the eyelids. A 1 to 1,000 solution of mercury bichloride has much support for its use in the treatment of folliculitis and also as an ingredient of scalp lotions.

In the treatment of sycosis vulgaris an occasionally more effective antiseptic is quinolor® (chlorohydroxyquinoline). At times, however, its employment is followed by a dermatitis. Drugs that are also excellent in the treatment of folliculitis, impetigo, ecthyma and other pyodermas are 1 per cent to 3 per cent ointment of iodochlorohydroxyquinoline (vioform®) and 3 per cent to 10 per cent ointment of bismuth tribromo-

phenate (xeroform®). Dermatitis from the latter two is uncommon, and their employment generally brings gratifying results. With rare exceptions it should not be necessary for one to use preparations of the sulfonamide drugs or penicillin topically in the treatment of cutaneous infections. These preparations are seldom as effective topically as the drugs previously mentioned, and their use in this form is hazardous.

Sulfur has an old and honored place in dermatologic therapy. For seborrheic dermatitis it still is the most effective medication, either alone in a 3 per cent to 5 per cent ointment or with the addition of 1 per cent to 3 per cent salicylic acid. In various sulfur lotions, such as white lotion, sulfurated lime solution or Kummerfeld lotion (a compound containing camphor spirit, alcohol, tragacanth, precipitated sulfur and distilled water), it has retained its popularity in the treatment of acne. Scabies may still be cured by the application of sulfur in a 3 to 10 per cent ointment. A 15 per cent solution of sodium thiosulfate will cure tinea versicolor without any messing. The pruritus of dermatitis herpetiformis is often relieved by application of a 2 per cent sulfur ointment. In the form of a 30 or 40 per cent sulfur paste, sulfur is frequently beneficial in the therapy of seborrheic dermatitis, dermatitis herpetiformis, chronic eczema and other chronic dermatoses.

Tar is a remedy of great value for many dermatoses. In nummular eczema and infantile eczema coal tar ointment generally produces rapid improvement, and in eczema of the nipple the response is excellent. Tar ointments are beneficial in treatment of tineal infection of the hands and feet and at times also in that of psoriasis and recalcitrant eruption. Solutions of crude coal tar in acetone, chloroform or collodion are much cleaner to apply but are not as effective as the ointment form. The various distilled or decolorized tars are also much less effective than crude coal tar, but distarol® and zetar® are comparatively good clean substitutes. The wood tars (pine tar, juniper tar and rectified oil of birch tar) should be used only in chronic dermatoses, for their employment in more acute stages generally increases the inflammation. "Oil of cadeberry"[1] has all the benefits of juniper tar, but it has a much more pleasant odor. In treatment of resistant patches of psoriasis or lichen chronicus simplex, tar-containing ointments, such as compound ointment of sulfur or Dreuw's ointment (a preparation containing salicylic acid, chrysarobin, rectified oil of birch tar, medicinal soft soap and petrolatum) may be used. Tar should never be used in any form in the presence of furuncles or other pustular infections, for fear of the production of lymphangitis. Folliculitis from the use of tar can generally be prevented by removal with a bland oil of all the old residue on the skin before a new application is made.

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1. A product of Muth Bros. & Co., Baltimore, containing barbados tar and oil of juniper berry.

Naphthalan and ichthammol (ichthyol®) in 5 to 10 per cent ointments are often helpful in the treatment of many chronic dermatoses.

Chrysarobin in a 2 to 10 per cent ointment is useful in the treating of chronic patches of psoriasis and lichen chronicus simplex, while a 4 per cent suspension in chloroform works well in the treatment of chronic paronychia and tineal infections between the toes. The drug is sometimes extremely irritating, and it is generally being replaced by anthralin ointment (1/10 to 1 per cent). Other drugs with similar action are pyrogallol, acetpyrogall (triacetyl pyrogallol; lenigallol®), anthrarobin and cignolin.

Resorcinol is widely used in combination with sulfur lotions in treatment of acne and also in treatment of seborrheic dermatitis. For use on the scalp it is available as resorcinol monoacetate (euresol®).

Salicylic acid is added to many ointments for its keratolytic effect. It is an active fungicide, and in benzoic and salicylic acid ointment it probably is still the most effective medication for ringworm infections. Thymol may be added (0.25 per cent) to increase the fungicidal activity. Fifty per cent salicylic acid in petrolatum has been recommended in the treatment of kerion, but in my experience milder ointments have been as effective. Forty per cent salicylic acid plaster is effective in the removal of calluses and corns.

Silver compounds are drugs that deserve wider use than they enjoy at present. Wet dressings of silver nitrate solution (in a dilution of 1 to 1,000 or 1 to 500) often produce a spectacular drying of acute, weeping streptococcic infections on the legs and behind the ears. Ten to 20 per cent solutions are useful for cauterizing fissures, and I have found daily applications of 10 per cent silver nitrate solution extremely helpful in treating ingrown toe nails. The granulation tissue is destroyed in these cases and is replaced by a tough crust that resists the pressure of the toe nail. In treatment of seborrheic dermatitis of the eyelids a 5 per cent solution of argyrol® (a combination of silver with a protein produced by the electrolysis of serum albumin) dropped into the eye is often helpful. Chronic ulcers are effectively treated by the Mikulicz ointment, which contains 1 per cent silver nitrate and 10 per cent Peruvian balsam in petrolatum.

Iodine in the form of a 10 per cent ointment in lard in my experience is more efficacious in treatment of ringworm of the scalp than are the newer fatty acids. Another old remedy for this disease is a 3 per cent solution of salicylic acid in tincture of iodine. In alopecia areata 3 per cent iodine in petroleum benzine has the advantage of staining the skin less than does tincture of iodine or Cutler's fluid (equal parts of liquified phenol tincture of iodine and chloral hydrate). In this disease local applications of phenol, soon removed with alcohol, also act as an adequate irritant.

Copper in the form of a 10 per cent ointment of copper oleate is therapeutically active in ringworm of the scalp, and alibour water (1 to 20) is an excellent wet dressing for Bockhardt's impetigo and other infected lesions.

It is possible that hexachlorocyclohexane in a vanishing cream base (kwell® ointment) will replace all other antiparasitic drugs. Until its place has been firmly established and its possible dangers are fully understood, it is well to remember that other good remedies for scabies include sulfur; Peruvian balsam and its derivative, benzyl benzoate; betanaphthol, and storax.

Benzoin tincture is often applied to fissures. A 50 per cent ointment of benzoin tincture is an excellent remedy for chronic ulcers, as is the application of Aloe vera leaf or ointment. Urea solution is useful in removing necrotic tissue from ulcers. Cod liver oil ointments are efficacious in the treatment of chronic ulcers and of burns, and for burns trinitrophenol solution is rapidly effective in treatment of patients who can tolerate it.

Of the various dyes used for topical application, Castellani's paint (a preparation containing basic fuchsin, phenol, boric acid, acetone and resorcinol) is by far the most valuable in the treatment of tineal infections on moist areas. It is best to begin by using a 25 per cent strength paint and then to strengthen it gradually. A 1 per cent solution of methylrosaniline chloride is excellent in treatment of yeast infections, especially those on the webs of the fingers and in the folds of the skin and paronychia. A 5 per cent solution of methylrosaniline chloride in 20 per cent alcohol is effective in the prevention of infection at the site of removal of warts or moles. Five per cent scarlet red ointment is an old and effective remedy for chronic ulcers.

For screening agents in lupus erythematosus and sensitivity to light, preparations of phenyl salicylate, quinine or tannic acid are satisfactory and are less likely to produce dermatitis than sodium para-aminobenzoate (Paba®) cream.

The application of 10 per cent bergamot oil in alcohol followed by exposure to the ultraviolet rays at times hastens the return of pigment in patches of vitiligo.

Five per cent ethyl aminobenzoate (benzocaine®) in flexible collodion applied to insect bites relieves the pruritus in many cases.

Condyloma acuminatum is best treated by the application of 25 per cent podophyllum resin in alcohol. One must exercise great care not to treat too large an area at one time, for fear of producing severe inflammation.

Verrucae under the nails or about the nails may be treated by applying 4 per cent formaldehyde solution, sulfurated lime solution or mono-

chloroacetic acid. Applications of trichloroacetic acid remove patches of xanthelasma. Monochloroacetic acid may be used in destroying seborrheic verrucae. After the Sherwell removal of an epithelioma solution of mercuric nitrate or ferric subsulfate solution may be applied.

For Vincent's infection of the mouth the administration of penicillin troches is no doubt the most effective local treatment. It is a mistake, nevertheless, for one to form the habit of using penicillin routinely in all oral conditions. In most of them nothing is accomplished by penicillin that cannot be performed as well and more safely by mild mouth washes, such as sodium bicarbonate, magnesia magma or the N. F. antiseptic solution. As a local astringent a mixture of kino tincture and myrrh tincture has a well founded reputation.

Finally, for the treatment of itchy legs nothing brings relief as rapidly as the application of Unna's paste boot.

Many additional drugs have been recommended for the treatment of various dermatoses. I have excluded them from this listing either because I did not have personal experience with their use or because they duplicated without advantage other drugs that are more readily available in the average pharmacy.

COMMENT

I feel certain that the medications listed have found great success in the work of some dermatologists and have been complete failures with others. The fault is not entirely with the drug, for each drug is but an inanimate servant, capable only of performing the limited tasks within its scope. The selection of the particular drug and the determination of the proper concentration, the ideal vehicle and the best method of application are often the factors that decide the therapeutic results. In addition, the unmeasured ingredient of every medication is the attitude of the physician who prescribes it. Some dermatologists have the power to inspire confidence and to obtain from the patient full cooperation in the carrying out of carefully outlined instructions. For one to tell a patient merely to put on cold wet dressings, for example, is to assume that the descriptive command means to him the same as it does to the physician. A patient is more likely to perform his duties correctly if he is permitted to explain to the physician just how he plans to carry out the orders—and is then corrected. Only by making certain that the patient is going to use the medications as prescribed can one hope to obtain the anticipated results. This added point may seem trivial to some, but one may also expect much better results from medication prescribed with an air of confidence than from that given with a skeptical attitude that almost dares the patient to get well.

I should not like to be set down as an obstinate reactionary, intolerant of all that is new. Certainly the sulfonamide compounds, antibiotics,

antihistaminic agents and other new drugs have opened new roads to hope in the treatment of many diseases hitherto serious in their prognosis. In many instances more progress can be made in hours with the use of new drugs than was possible in months with old-fashioned treatment. The risk of sensitization and toxicity inherent in many of these drugs makes it prudent, however, that they be used only when they are needed. Not only dermatologists, but also physicians lacking dermatologic training, have tended too much in recent years to try these new drugs as the first medication in the treatment of all dermatoses before turning to older drugs that have proved adequate. Furthermore, because of the vital importance of sulfonamide compounds and antibiotics in the treatment of dangerous infectious diseases, it is imperative that these drugs be reserved, if possible, for exigencies when they are desperately needed. We dermatologists are seldom called on to treat diseases so serious as to warrant the risk of exhausting a patient's capacity for using a drug that might in the future prove to be life saving.

A dissertation on good dermatologic therapy utilizing well established drugs would be incomplete if it failed to stress the most important requisites for obtaining good therapeutic results, namely the correct diagnosis of the disease and the proper appraisal of the stage of activity present. One must recognize, for example, not merely that he is dealing with a tineal infection of the feet before instituting proper treatment; it is just as important for one to ascertain whether therapy should be actively fungicidal, in the chronic stage of the disease, or merely soothing, when the skin is acutely inflamed. It is important for the physician to recognize the clinical picture of overtreatment dermatitis that may be masking the features of the original dermatosis. Discontinuing all previous medication in such cases and the cautious use of soothing treatment will often give the patient great relief and may also bring to light the diagnostic features present. One should, in addition, always keep in mind that he is treating a patient with a cutaneous disorder rather than merely treating a cutaneous disease. All of us need constant reminding that the skin is often a window to internal disease. A dermatologist's diagnostic eye and therapeutic acumen should encompass not only the skin but also the patient living within it.

Finally, a clinical dermatologist's chief function (and most of us are primarily clinicians) is to bring to his patients relief from suffering. To this end he should utilize all measures of established merit that apply to each case. For this vocation most of us are well trained. It has become popular in recent years, however, for many dermatologists to become clinical investigators for every new drug and proprietary mixture that is brought to market. For this highly critical function most of us are not adequately equipped. Not many have a large enough

group of patients to investigate fully the effects of a drug, with the controls essential to an accurate appraisal. Many clinical investigators as yet lack the mature judgment needed to temper their enthusiasms and to keep them from being carried away by clinical impressions that may not be lasting. In dermatologic therapy too many are attempting to run who have not yet fully mastered the art of walking. It may seem antiquated to use old drugs and old fashioned to treat patients with the simple purpose of getting them well as quickly as possible. Certainly there is more glamour in being up to the minute with the use of the newest drugs and even ahead of the times in the investigation of drugs for which no specific use has yet been found. Most of us dermatologists, in my opinion, are most useful, however, when we devote ourselves wholeheartedly to the career of being healers of the sick. It is an old-fashioned role, devoted largely to treating diseases that are not new and most often calling for the employment of drugs that have been tested in the crucible of time. It is an honorable career and a satisfying one, a career to which, in our highly scientific age, we particularly need converts.

25 East Washington Street.

SPECIFIC SENSITIVITY TO FOODS AS A FACTOR IN VARIOUS TYPES OF ECZEMATOUS DERMATITIS

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MANY observers[1] have reported cases of chronic, recurrent, vesicular eruptions of the hands due to various food allergens. Other authors,[2] who have written on the problem of eczematous dermatitis of the hands, designated by various terms such as "chronic eczematoid dermatitis of the hands" and "recurrent vesicular eruptions of the hands," have listed food allergy as one of the infrequent and relatively unimportant etiologic factors. Stokes,[3] in particular, emphasized the concept that such eruptions of the hands, as well as chronic eczematous dermatitis at other sites, are often due to multiple etiologic factors. He stressed the importance of a "factorial analysis" in the study of such patients and included food allergy among the various etiologic considerations.

Read at the Sixty-Eighth Annual Meeting of the American Dermatological Association, Inc., San Diego, Calif., April 27, 1948.

From the Department of Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania, Dr. Donald M. Pillsbury, Director.

For many of these patients, scratch, intradermal and patch tests were done by Dr. Norman R. Ingraham Jr. Our associate, Dr. Malcolm C. Spencer, assisted in the management of some of the patients included in this series.

1. Buckley, L. D.: Diet and Hygiene in Diseases of the Skin, J. A. M. A. **59**:535 (Aug. 17) 1912. Wise, F., and Wolf, J.: Dermatophytosis and Dermatophytids, with Particular Reference to Differential Diagnosis of Dyshidrosiform Eruptions of Hands and Feet, Arch. Dermat. & Syph. **34**:1 (July) 1936. Wise, F., in discussion on Lehmann, C. F.: Acute Vesicular Eruptions of the Hands and Feet, ibid. **21**:499 (March) 1930. Engman, M. F., Jr., in discussion on Andrews, G. C.; Berkman, F. W., and Kelly, R. J.: Recalcitrant Pustular Eruptions of Palms and Soles, ibid. **29**:548 (April) 1934.

2. Lane, C. G.; Rockwood, E. M.; Sawyer, C. S., and Blank, I. H.: Dermatoses of the Hands, J. A. M. A. **128**:987 (Aug. 4) 1945. Callaway, J. L., and Barefoot, S. W.: The Diagnosis and Management of Chronic Eczematoid Dermatitis of the Hands, North Carolina M. J. **1**:547, 1940.

3. Stokes, J. L.; Lee, W. E., and Johnson, H. M.: Contact, Contact-Infective and Infective Allergic Dermatitis of the Hands, with Especial Reference to Rubber Glove Dermatitis, J. A. M. A. **123**:195 (Sept. 25) 1943.

In 1946, Rowe [4] reported a series of 80 patients (42 per cent of 182 patients with eruptions of the hands) with "atopic dermatitis of the hands" due to food allergy, and concluded from his observations that "dermatitis of the hands in the average population may result more frequently from atopic allergy to food than from any other cause."

At about the same time, Flood and Perry [5] reported two series of patients, 30 patients with "recurrent, vesicular eruptions of the hands" being discussed in one paper,[5a] and 13 patients with "eczematoid dermatitis" in the other,[5b] with food sensitivity comprising the primary etiologic factor. Flood and Perry did not attempt to estimate the exact incidence of food allergy as the predominant factor in such eruptions, nor did they compare its importance with other factors, but they implied that specific sensitivity to foods is at least as important as any other etiologic factor.

Our experience in the study and treatment of eczematous dermatitis of the hands, feet and other sites is recorded in this paper with an attempt at a critical evaluation of the relative importance of specific food sensitivity as a primary or contributory etiologic factor in such eruptions. One of us (C. S. L.) had an opportunity to accumulate considerable experience in this therapeutic approach during World War II, but none of the patients encountered at that time is included in this report because adequate follow-up was not possible. All the patients in this series were seen and followed in our joint private practice. We have included only those patients with an adequate period of follow-up observation and in whom a predominant role of food allergy in the eruption seemed inescapable. Furthermore, we have not included patients from whom complete cooperation was not obtained. This fact has limited the number of patients in our series, but it is believed that the experience with this group is representative and the major aspects of this subject are illustrated in the case material. It is believed that one reason for some difference of opinion in regard to the importance of the food allergy factor in eczematous dermatitis is the failure to realize that food sensitivity is only one of the etiologic agents in many of these cases and that the complete cure and, indeed, recognition of a contributory food allergy factor depend on the identification and adequate management of all the factors concerned in the individual case. This principle is well illustrated in many of our cases.

It is essential to attempt to clarify the descriptive terms applied to this group of eczematous eruptions because nomenclature is cumbersome

4. Rowe, A. H.: Atopic Dermatitis of the Hands Due to Food Allergy, Arch. Dermat. & Syph. **54**:683 (Dec.) 1946.

5. (*a*) Flood, J. M., and Perry, D. J.: Recurrent Vesicular Eruptions of the Hands Due to Food Allergy, J. Invest. Dermat. **7**:309, 1946; (*b*) The Role of Food Allergy in Eczematoid Dermatitis, Arch. Dermat. & Syph. **55**:493 (April) 1947.

and confusing, especially for the physician who is not a dermatologist. The same type of disease process has been referred to by various authors as eczema (with numerous qualifying terms such as vesicular and nummular), eczematoid dermatitis of the hands and feet, chronic, vesicular eruptions of the hands, "atopic dermatitis of the hands" and "eczematous contact-type allergic dermatitis," among other terms. It would be desirable to use terms based entirely on etiology, but this is not possible because the cause in many cases is unknown; in other instances the etiologic agents are multiple, and they may vary from time to time in the same patient. Therefore, any classification must be partly etiologic and partly morphologic. There is general agreement in the use of the following terms (or various synonyms), either because the dermatitis is due to a single etiologic factor or because of the characteristic type, distribution and course of the cutaneous eruption: (1) contact dermatitis, (2) dermatitis medicamentosa (eruptions due to drugs), (3) atopic dermatitis, (4) infantile eczema of atopic type, (5) seborrheic dermatitis and (6) lichen simplex chronicus.

We use the term eczematous dermatitis as a morphologic diagnostic term for all other types of chronic and recurrent dermatitis due to a wide variety of external and internal causes or of unknown etiology. It is the group of cases in this category which has been responsible for most of the confusing synonyms. The individual entities within the syndrome are often not sharply defined, and transitional types are encountered. For example, it may be difficult to determine whether the patient has a contact dermatitis with secondary bacterial infection, or a pyoderma with secondary eczematous changes due to sensitization to bacteria, or reactions to local treatment.

The cataloging of the individual patient into a distinct diagnostic category depends on qualification of the term by adding the site or sites of involvement and the etiologic factors, if they are determined and proved (so far as doing so is possible). The following etiologic factors are variously recognizable in (chronic) eczematous dermatitis:

1. Allergic contactant
2. Primary irritant

(Dermatitis in which these factors are the only factors is classified under the heading of contact dermatitis.)

3. Food allergy
4. Secondary bacterial invasion and sensitization (Impetigo and other primary uncomplicated pyogenic infections of the skin are not included.)
5. Drug allergy (If this is the only factor the condition should be included under the heading of dermatitis medicamentosa.)
6. Pollen and inhalant allergy
7. Mycotic infection and mycotic sensitization (Uncomplicated primary fungous infection and dermatophytids are classified as such.)
8. Psychosomatic, traumatic, thermal, endocrine, peripheral, vascular and dyshydrotic factors and foci of infection
9. Undetermined etiologic factors

It is realized that there are other possible contributory factors, such as physical allergies, diseases of the gastrointestinal tract, liver or kidney, intercurrent infections of various types and metabolic disturbances.

METHOD OF STUDY

It is our practice to consider specific food sensitivity as a possible primary or contributory factor in all cases of eczematous dermatitis, regardless of the site of involvement, unless it is obvious on initial examination or a brief subsequent period of observation that other causes are responsible.

The methods of study in regard to a possible food allergy factor which we have used include one or more of the following regimens:

(1) A strict trial diet (as outlined by Flood and Perry,[5]) which is feasible only for hospitalized patients

(2) A so-called basic "nonallergenic diet," consisting of beef, lamb, chicken, canned pears, canned apricots, prunes, baked or boiled potatoes, lettuce, celery, string beans, red beets, lima beans, weak tea, sugar, salt, white vinegar, ry-krisp® or pure rye bread, peach preserves and apple jelly, followed by addition of foods one at a time at five-day intervals after a base line of improvement has been established

(Recently, following the suggestions of Winston and Sutton,[6] we have added only one food at each meal. [For example, on the first day of the diet, the menu is as follows: Breakfast—canned pears; luncheon—canned pears and tea with sugar, and dinner—canned pears, tea with sugar and lamb. On subsequent days other foods of the basic diet are added in a similar manner.] If pruritus and/or new lesions of any type are noted after the addition of a new food, all new foods which have been added in the previous twelve-hour period are eliminated from the diet.)

(3) A minimal elimination diet ("spot" elimination diet), interdicting sea foods, chocolate, nuts, citrus fruits, tomatoes, pork, cheese and coffee

(4) A diet diary

It must be realized that considerable individualization is necessary, inasmuch as there are practical limitations in the study and treatment of patients from the standpoint of food allergy. A high percentage of patients do not like strict diets; hospitalization is not available or feasible for everyone, and, on an outpatient basis, business and social responsibilities make it difficult for persons to follow even simple elimination diets. There are very few patients who find it possible to eat all their meals at home, and, if they do, the necessity of special cooking for one member of the family is a complicating factor. Furthermore, the vast majority of patients are not easily convinced that food allergy may constitute a factor in their particular case. Only a small number of our patients will follow diets as limited as the Rowe elimination diet[6] or the so-called "basic nonallergenic" diet for more than two

6. Winston, B. H., and Sutton, R. L., Jr.: Dermatitis of the Hands Due to Ingested Allergens, Arch. Dermat. & Syph. **58**:335 (Sept.) 1948.

weeks, and, if there is no significant improvement at the end of that time, there is difficulty in persuading the patient to continue any type of dietary restriction.

The evaluation of the course of the eruption is complicated by the fact that the interval between the ingestion of food and the beginning of the exacerbation varies in different patients. Furthermore, there is an important quantitative factor, and, in addition, the presence of concomitant pyogenic and pyogenic sensitization, allergic contactant or other factors interferes with the interpretation of results.

It was shown by many investigators [7] that limitation of the diet on the basis of scratch tests to food is rarely helpful, and our experience is in agreement with this conclusion. However, many patients who do not like dietary restrictions in principle will cheerfully adhere to a restricted diet based on the results of scratch tests, often for years.

Because of all these limitations, we have been obliged to use various methods of approach, including the strict trial diet as outlined by Flood and Perry, a basic nonallergenic diet which follows the general principles of the Rowe elimination diets and a "spot" elimination diet from which foods with a high allergenic index are eliminated. All the patients who were studied by means of the strict trial diet were hospitalized. After hospitalization, during which pronounced improvement or complete cure was affected in all patients in whom food allergy was an important etiologic factor, new foods were added at five day intervals. Diet diaries were used in all cases because, for one thing, they assist patients in learning to recognize their exacerbations and to correlate them with the addition of possible causative foods.

PRESENTATION OF CASES

We have been able to demonstrate either a primary etiologic or an important contributory etiologic food factor in a total of 26 patients who have had adequate follow-up observation. For an additional 41 patients, specific sensitivity to food was an apparent important component of the dermatitis, but we were not able to establish unequivocal proof of this fact, or the observation period was not sufficient at the time of writing, and, therefore, their cases are not included in the series which constitutes the basis for our discussion.

Lack of space makes it impossible for us to include the individual case histories of all these 26 patients. Therefore, only 3 representative cases are presented in detail; pertinent data on all the 26 patients are summarized in the accompanying table.

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7. (*a*) Winston and Sutton.[6] (*b*) Rowe, A. H.: Elimination Diet and the Patient's Allergies: A Handbook of Allergy, ed. 2, Philadelphia, Lea & Febiger, 1944.

REPORT OF CASES

CASE 1.—M. C., a housewife aged 32, was seen because of a patchy, eczematous dermatitis of twenty years' duration, involving the hands and the left lower eyelid, the eyelid having become involved only within the past four months. The eruption was characterized by erythematous, scaling, oozing, rather well demarcated, vesicular plaques on the right palm and on all surfaces of the right third finger, as well as on the sides and dorsal surface of the second and fifth fingers of the left hand. The patient had severe pruritus, which had interfered with sleep and other activities. Several weeks after she came under our observation, she also had a brief episode of an oozing, pruritic, intertriginous dermatitis of the axilla and groins. At various times vesicopustules and residual pyognic crusts were noted as a part of the process. This patient had not been completely free of lesions since the onset, although there had been many exacerbations and partial remissions. The longest and most significant partial remission had occurred approximately two years before her first visit, during the time that she was on a reducing diet consisting of grapefruit juice, eggs, toast, cheese, tea, lettuce and tomatoes. On several occasions during the past three years, she had had attacks of intertriginous dermatitis in the axilla and groins persisting for two to three weeks, followed by a complete remission.

The patient had been treated by at least twelve different physicians, including five dermatologists, who had given her a total of approximately twenty-five roentgen ray treatments, and she had been informed by one of her physicians that he had given her as much roentgen therapy as her hands would tolerate. The use of numerous ointments and other types of local treatment, as well as the employment of a soap substitute and strict avoidance of soap and water and all household contactants, had had no apparent effect on the course of her lesions.

There was a history of infantile eczema in early infancy and of relatively transient attacks of acute urticaria in childhood. The familial allergic history was noncontributory except that the patient's sister had had apparent hydrorrhea due to vasomotor neurosis (vasomotor rhinitis) and urticaria.

The management and course of this patient's eczematous dermatitis during the eighteen months of observation were as follows:

1. Patch tests with numerous household contactants, cosmetics and routine test materials had negative results except for 1 plus reactions to dilutions of 1:1,000 of bichloride of mercury, prell® shampoo, lustre creme® shampoo, rinso® and resorcin, although all these reactions were minimal.

2. Initially, on an outpatient basis, the patient was placed on a diet of baked potato, beef, lamb, string beans, carrots, tea, sugar, grapefruit juice, boiled eggs, canned pears, prunes, canned apricots, lettuce and tomatoes. In addition, she was asked to avoid prell® shampoo, lustre creme® shampoo, and rinso.® Local treatment consisted of potassium permanganate soaks and 3 per cent ichthammol (ichthyol®) in zinc oxide ointment. Housework of all types was reduced to a minimum by the patient's employing a full time maid and enlisting the cooperation of her husband for dishwashing when the maid was not available. She did not have significant, sustained improvement on this regimen of treatment.

3. Therefore, she was hospitalized for a period of two weeks. She was placed on a strict trial diet (Flood and Perry), with sugar water during the first twenty-four hours, baked potato during the next twenty-four hours and lamb during the next twenty-four hours; during this time there was pronounced continued improvement, with almost complete disappearance of pruritus after the first forty-eight hours. Tea was added on the fourth day, and eight hours after the first cup of tea,

Data Concerning Twenty-Six Patients with Eczematous Dermatitis

Case No.	Age	Sex	Occupation	Duration	Period of Observation	Sites of Involvement	Methods of Study	Factors	Foods Which Caused Exacerbation	Comment
1	32	F	Housewife	20 yr.	18 mo.	Hands, left lower eyelid, axillas, groins	Hospitalization, strict trial diet, diet diary	1. Food allergy 2. Pyogenic infection and pyogenic sensitization	Tea, apricots, broccoli, cauliflower, cabbage, peaches	Pyogenic factor treated with penicillin administered parenterally
2	29	F	Nurse and housewife	1 yr.	6 mo.	Hands, left lower eyelid	Basic "nonallergenic" diet, diet diary	1. Food allergy 2. Contactant	Eggs, milk	Onset as contact dermatitis
3	30	F	Secretary	15 mo.	1 yr.	Feet, hands	Basic "nonallergenic" diet, diet diary	1. Food allergy	Wheat, chocolate, coffee, shrimp, crab, lobster	Scratch test-negative diet partially successful
4	28	M	Mail carrier	4½ yr.	9 mo.	Hands	Hospitalization, strict trial diet, diet diary	1. Food allergy 2. Pyogenic infection and pyogenic sensitization	Milk, chocolate, pork, tomatoes	Pyogenic factor treated with parenterally administered penicillin combined with fever therapy
5	44	M	School teacher	1 yr.	3 mo.	Perianal region, scrotum, hands	Spot elimination diet, diet diary	1. Food allergy	Chocolate, shad, shrimp	No resemblance of scrotal dermatitis to lichen simplex chronicus
6	30	F	Secretary and housewife	6 mo.	4 mo.	Hands	Basic "nonallergenic" diet, diet diary	1. Food allergy	Cinnamon, onions, eggs, smoked ham	
7	55	F	Housewife	6 mo.	2 yr.	Hands	Spot elimination diet, diet diary	1. Food allergy 2. Contactant	Nuts, grapefruit, oranges	Prominent psychogenic factor
8	27	M	Medical student	2 mo.	16 mo.	Hands	Hospitalization, strict trial diet, diet diary	1. Food allergy 2. Contactant 3. Drug	Wheat, milk, pork(?), chicken(?), carrots(?)	Exacerbations with use of naphazoline ("privine") nose drops; patient not cured until smoking was discontinued; positive reaction to patch test with tobacco
9	67	M	Civil engineer	1 yr.	17 mo.	Legs, ankles, thighs, hands, left upper and lower eyelids	Basic "nonallergenic" diet, diet diary	1. Food allergy	Wheat, veal, rye	No improvement until responsible foods were discontinued for several weeks—which observation is unusual
10	37	F	Housewife	6 yr.	20 mo.	Hands	Hospitalization, strict trial diet, diet diary	1. Food allergy 2. Pyogenic infection and pyogenic sensitization 3. Drug	Broccoli, string beans, lima beans, vanilla, peas, sweet corn	Exacerbation on ingestion of sulfadiazine and pentobarbital sodium, acetylsalicylic acid; pyogenic factor treated with penicillin given parenterally
11	31	F	Housewife	4 mo.	9 mo.	Hands, legs	Basic "nonallergenic" diet, diet diary	1. Food allergy	Wheat, chocolate, peaches(?), pork(?)	Unusually severe, extensive involvement, with rapid improvement on management on an outpatient basis
12	30	F	Secretary	5 mo.	18 mo.	Hands	Spot elimination diet, diet diary	1. Food allergy	Grapefruit, peanuts, oranges	Prominent psychogenic factor
13	4	M	Preschool child	1 yr.	6 mo.	Buttocks, legs	Basic "nonallergenic" diet, diet diary	1. Food allergy	Eggs, chocolate, pork	Exacerbations on numerous attempts to add causative foods to diet
14	50	M	Executive	1 yr.	1 yr.	Hands, legs	Hospitalization, strict trial diet, diet diary	1. Food allergy	Almonds, tomatoes, pork, Scotch whisky, oranges(?)	Patient completely incapacitated by his dermatitis

15	56	F	Secretary	1 mo.	1 yr.	Hands, forearms, neck, right upper eyelid	Basic "nonallergenic" diet, diet diary	1. Food allergy	Oranges, grapefruit, wheat, coffee(?), eggs(?), pineapple(?)	Prominent psychogenic factor
16	56	F	Secretary	10 yr.	6 mo.	Right upper eyelid, right postauricular area	Spot elimination diet, diet diary	1. Food allergy	Chocolate, lobster, clams, crab	Exacerbations following ingestion of food, accompanied with nausea and gastrointestinal pain
17	22	F	Salesgirl	11 yr.	8 mo.	Hands	Scratch test-negative diet, diet diary	1. Food allergy	Cheese, pimento, pork, pecans	The only patient in our series for whom a scratch test-negative diet was successful; happened to result in the elimination of all causative foods
18	12	M	Student	3 mo.	1 yr.	Posterior surface of neck	Spot elimination diet, diet diary	1. Food allergy	Chocolate	Repeated exacerbations on addition of chocolate to diet; after six months patient able to eat chocolate occasionally without exacerbation
19	52	M	Executive	6 mo.	8 mo.	Hands	Basic elimination diet, diet diary	1. Food allergy 2. Pyogenic infection	Rice	Clearing of bacterial infection along with improvement of dermatitis, without specific antibacterial therapy
20	21	F	Secretary	15 mo.	6 mo.	Hands, right cheek	Basic elimination diet, diet diary	1. Food allergy	Milk, wheat	Repeated sharp exacerbations on addition of milk or wheat products to the diet
21	40	M	Clerk	4 yr.	6 mo.	Hands	Hospitalization, strict trial diet, diet diary	1. Food allergy 2. Pyogenic infection and pyogenic sensitization	Chocolate, peanut butter	Incomplete clearing until pyogenic factor had been treated with parenterally administered penicillin and fever therapy
22	39	F	Housewife	4 yr.	2 yr.	Hands, feet	Hospitalization, strict trial diet, diet diary	1. Food allergy 2. Pyogenic infection and pyogenic sensitization 3. Contactant	Pepper, cinnamon, chicken, bacon, smoked ham, onions, paprika	Persistence of sensitivity to causative foods for as long as eighteen months after clearing of eruption
23	28	F	Secretary	3 yr.	1 yr.	Face	Hospitalization, strict trial diet, diet diary	1. Food allergy 2. Drug	Rice, butter, sweet potatoes, carrots, tomatoes, oranges, eggs, chocolate	Eczematous and folliculopustular lesions; repeated exacerbations after ingestion of vitamin B complex capsules
24	20	F	Nurse	2½ yr.	2 yr.	Left axilla	Hospitalization, strict trial diet, diet diary	1. Food allergy 2. Contactant	Oranges, grapefruit	Eruption resembling a fixed drug eruption
25	42	M	Physician	3½ yr.	6 mo.	Hands	Hospitalization, strict trial diet, diet diary	1. Food allergy 2. Contactant 3. Drug	Tomatoes, oranges, grapefruit	Phenobarbital, both as contactant and ingestant, one of the etiologic factors
26	28	M	Foundry worker	3 yr.	9 mo.	Hands, ankles	Hospitalization, strict trial diet, diet diary	1. Food allergy	Pork, milk, potatoes	Onset of dermatitis, on the dorsal surface of the hands, as a contact dermatitis due to a primary irritant; persistence for years (five) and complete disappearance on elimination of causative foods; repeated exacerbations demonstrated on addition of responsible foods; flare-ups within fifteen to thirty minutes after ingestion of causative food

an interval during which the patient had had 3 cups, there was a sharp, distinct, relatively severe exacerbation, characterized by intense pruritus and recurrence of erythema, vesiculation and oozing. Within a brief period evidence of secondary bacterial infection was noted. After this episode, 50,000 units of penicillin every three hours was administered for five days, during which time there was a slow but continued improvement. At the end of two weeks of hospitalization, the eczematous dermatitis was less active than at any other time since the patient had come under our observation. There were no vesicles or pustules, and the pruritus had almost disappeared. However, there was some residual scaling, erythema and superficial fissuring. The patient was discharged from the hospital, with instructions to follow a diet consisting only of baked potato, lamb and string beans.

4. Continued improvement was noted during the next seven weeks except for significant exacerbations following the trial addition of tea and dried apricots to the patient's diet. During this time, the diet had been increased to include beef, lamb, string beans, sugar, chicken, turkey, lettuce, asparagus, carrots, baked potatoes, canned pears, prunes, apples, eggs, white bread, saltines, vanilla ice cream, milk and ginger ale. All these foods had been added gradually, one at a time, with approximately two to three days between each addition. No attempt was made to add further foods during the next month, and during this time the lesions on the hands and eyelids cleared up completely.

5. In the next six weeks, in the same manner, the patient added cottage cheese, American cheese, almonds, corned beef, bananas and cherry gelatin, without any exacerbations. During this same period she had prompt exacerbations manifested by pruritus, erythema and a few tiny vesicles at previously involved sites, four hours after the addition of broccoli and cauliflower to her diet on two separate occasions.

6. During the next six weeks the patient continued to remain free of any signs of involvement except for a slight exacerbation twelve hours after the addition of cabbage and canned peaches to her diet on two separate occasions. In the meantime she had added celery, onions, green pepper, mushrooms, beets, mashed turnips, beef and calf liver and several types of fish, so that at this time the only foods restricted from her diet were tea, dried apricots, broccoli, peaches, cauliflower and cabbage.

7. During the past year she has been free of involvement and has been able to do all her own housework with no effort to protect her hands from excessive contact with soap and water and other cleansing agents.

It is interesting to note that tea has been the main beverage of this patient throughout her adult life because she does not like coffee and, furthermore, that she has always eaten dried apricots.

The diagnosis was eczematous dermatitis of the hands, left lower eyelid, axilla and groins, with food allergy (tea, apricots, broccoli, cauliflower, cabbage, and peaches), pyogenic infection and pyogenic sensitization as factors.

Comment.—This is an example of a difficult food allergy problem. It is probable that this patient would have been included in the group of those in whom treatment was a failure or, rather, that the food allergy factor would not have been recognizable if it had not been possible for her to be hospitalized and if her cooperation had not been unusually good. It may be noted that this patient reacted to several members of the cabbage family, as well as to related fruits. She is the only patient in our entire series for whom tea was recognized as a food allergen.

CASE 2.—B. M., a nurse aged 29, had first noted approximately one year previously what was described as a pruritic, vesicular patch of dermatitis on the dorsal surface of her left hand. It had occurred approximately ten hours after she had mixed penicillin solution, during which procedure a moderate amount of the concentrated solution had come into contact with the affected site. During the next three months she had had several recurrences of pruritic, vesicular dermatitis on the dorsal surface of the hand, with complete remission between these episodes. After this period, she had had more frequent attacks, as well as extension of involvement to the dorsal surface and sides of several fingers. Furthermore, the lesions persisted between attacks, and at times superficial fissuring, which interfered with the movement of her fingers, occurred.

Six months after the initial onset, all lesions cleared up completely during a three weeks' vacation, when the patient was away from her work as a clinic nurse. There was prompt recurrence when she returned to work. At this time she consulted a dermatologist, who found positive reactions to patch tests with penicillin; contact with this drug was discontinued. There was slight improvement, but complete clearing did not occur, and the patient continued to have periodic exacer-

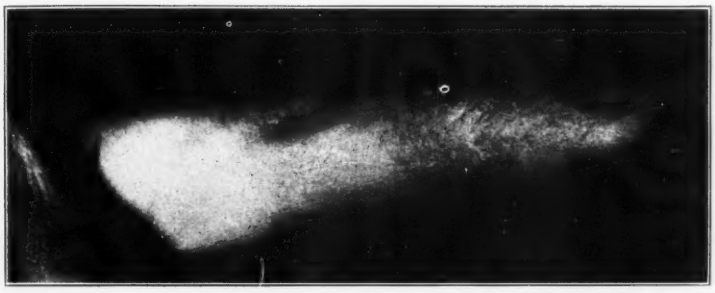

Fig. 1 (case 2).—One of the eczematous plaques on the fingers, which on one occasion flared up simultaneously with the exacerbation of a lesion on the eyelid.

bations. Patch-testing was then done with procaine hydrochloride (novocain®) and streptomycin, with positive results, and therefore contact with these drugs was discontinued. However, despite this action, during the next three months the patient continued to have repeated flare-ups, and finally it was necessary for her to discontinue her work for the second time. Within one week, there was significant improvement, but she continued to have new vesicles and also had one distinct, rather severe exacerbation which was certainly not related to contact with penicillin, procaine hydrochloride or streptomycin. At the time of the exacerbation she consulted an allergist, who confirmed the previously reported positive reactions to patch tests and also demonstrated that she had pronounced sensitivity to trichophytin; for this reason he advised a series of injections with trichophytin as an attempt at desensitization. Because of financial reasons, it was essential for her to return to work. There was no improvement, and indeed the severity and extent of her lesions gradually increased during the next two months; the exacerbations were not related to the injections of trichophytin.

Other treatment included strict avoidance of soap and water, with use of a soap substitute, superficial roentgen therapy and application of several unidentified ointments.

When the patient consulted us, she had seven distinct, rather well demarcated, erythematous, oozing, scaling, vesicular plaques on the dorsal surface and sides of three fingers of the right hand, two fingers of the left hand and the right palm. There was some crusting but no frank vesicopustules. In addition, she had superficial fissures in several plaques, and there was some limitation of movment of the involved fingers. The only other finding of importance was an erythematous, edematous, scaling, well demarcated plaque on the inner surface of the right upper eyelid. The latter lesion had been noted about two weeks previously.

Fig. 2 (case 22).—The exacerbation which is illustrated followed the ingestion of smoked pork. Concomitant bacterial infection was a complicating factor.

The familial and personal allergic history were noncontributory except that the patient had had what had been diagnosed as allergic vasorhinitis on one occasion when she was living in Texas. There was no history of any type of previous cutaneous disease or intolerance to any contactants encountered during her work as a nurse prior to the onset of her present trouble.

The management and course of this patient's eczematous dermatitis during the seven months' observation period were as follows:

1. She was advised to discontinue work, which she did for a three week period, and during this time Dr. Norman Ingraham Jr. did a complete allergy work-up,

a summary of which follows: Reactions to patch tests were negative for a long list of household and other contactants except for crystalline penicillin G (1 cc. containing 25,000 units), 3 plus; a 2 per cent aqueous solution of procaine hydrochloride, 3 plus, and o'cedar® furniture polish (50 per cent in olive oil), 3 plus. Scratch and intradermal tests with aqueous protein extracts of common dust-borne allergens, except for those with mixed feathers, sheep dander, rabbit hair and cow dander, had negative reactions. Scratch tests with 140 foods had negative results except for weakly to moderately positive reactions to almonds, bananas, lima beans, Roquefort cheese, cherry, chicory, cinnamon, coconut, dates, grapefruit, milk, mustard, onions and poppy seed.

2. She had about a 50 per cent improvement during the three week period when she was away from work, but she continued to have exacerbations characterized by intense pruritus and the appearance of new vesicles, particularly at the periphery of the involved sites. At times, the eyelid lesion flared up concomitantly with the

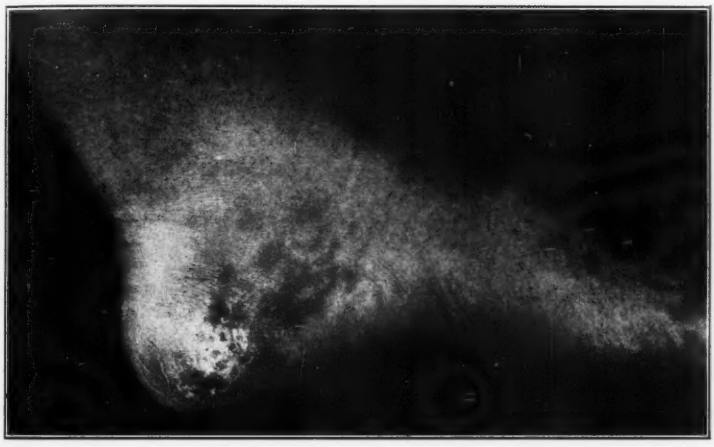

Fig. 3 (case 22).—This site of involvement had flare-ups on numerous occasions when one of the responsible food allergens was added to the diet. The lesions on the patient's hand are illustrated in figure 2.

lesions on her hands, but at other times either the eyelid lesion or the hand lesions flared up independently. The only local treatment which was used consisted of aluminum acetate solution (1:20) soaks several times daily, followed by application of 3 per cent itchthammol in zinc oxide ointment. After the allergy work-up, o'cedar® furniture polish was added to the list of contactants for her to avoid, and also the patient was asked to follow a diet consisting of only the foods which gave entirely negative reactions on scratch tests. She returned to work, inasmuch as the severity of the exacerbations and their frequency during the time when she was away from work were such that the financial sacrifice did not seem justifiable.

3. There was no significant improvement on a scratch test-negative diet during the next three week period, and therefore chocolate, coffee, orange, cheese and tomato and nuts were also eliminated from the diet; in addition, the patient was asked to keep a careful diet diary. Wheat and eggs were not added to the list

of foods to be eliminated at this time because it is most difficult for one to avoid these foods in a hospital dining room.

4. The patient had some improvement in her condition in the next two weeks, but continued to have periodic flare-ups, although they were less severe than previously. At this time wheat was added to the foods to be eliminated from the diet.

5. There was no change in the next week, and at this time it was decided to add eggs to the interdicted foods, which action meant that the total restriction of the diet included the elimination of almonds, bananas, lima beans, Roquefort cheese, cherries, cinnamon, chicory, coconut, dates, grapefruit, milk, mustard, onion and poppy seed, which had been eliminated on the basis of positive reactions to scratch testing, and chocolate, coffee, orange, cheese, tomatoes, nuts, wheat and eggs, which had been eliminated in an effort to obtain a so-called basic nonallergenic diet.

6. During the next two weeks, after the elimination of eggs from the diet, there was more sustained improvement than at any previous time since the patient had come under our observation, and for the first time there was complete absence of pruritus and no appearance of new vesicles within the last week.

7. Five days later the patient had a distinct flare-up at all previously involved sites, including the eyelid, characterized by pruritus, vesiculation, oozing, erythema and edema. This exacerbation occurred approximately six hours after she had eaten one boiled egg. It subsided entirely within five days.

8. After this development, the patient remained free of symptoms and lesions except for four exacerbations, all of which subsided promptly. The first such exacerbation did not occur until she had eaten one egg daily for three days; the second, until she had eaten one egg daily for six days, and the third, until after she had increased her intake of milk to one quart daily for three consecutive days. During the next six weeks all other interdicted foods were added, without any exacerbation, so that, at the end of this time, the restriction of her diet included only the elimination of eggs and the limitation of milk to one glass daily, with no restriction of foods containing milk.

9. During the next three months her hands and eyelid remained clear, despite the addition of all foods containing eggs to her diet, although she continued to restrict eggs to three weekly (one every other morning for breakfast). She had continued to work as a nurse and had done all her own housework, with avoidance of furniture polish, procaine hydrochloride, penicillin and streptomycin, but with no other restrictions or precautions.

The diagnosis was eczematous dermatitis of the hand and left eyelid, due to allergic contactants (penicillin, streptomycin, procaine hydrochloride and o'cedar® furniture polish) and food allergy (eggs and milk).

Comment.—It seems apparent that this patient originally had an allergic contact eczematous dermatitis and that the food allergy factor did not enter the picture until approximately six months after the onset of her dermatitis, which gradually increased in severity, presumably because of repeated exposure to the allergic contactants. It is obvious that the responsible foods would have been discovered promptly if this patient had been hospitalized or if a resrticted elimination diet had been put into effect early in our observation period. When egg was added to the diet one week after pronounced improvement had occurred, a

flare-up ensued within a short time after the ingestion of only one egg; six weeks after the lesions had become quiescent, a flare-up did not occur until the patient had eaten one egg daily for six consecutive days; several months later, she was able to eat three eggs weekly, as well as foods containing eggs, without any return of pruritus or objective signs. This spontaneous desensitization is a common sequence of events, provided that all causes of the eczematous dermatitis have been eliminated and the lesions have remained entirely clear for several weeks to several months. It may be noted that she had a negative reaction to a scratch test with the only significant food allergen which was one of the proved causative factors.

Fig. 4 (case 11).—The patient also had large, oozing, eczematous plaques on both legs. Numerous exacerbations such as the one illustrated were demonstrated on the addition of wheat and chocolate to the diet.

CASE 3.—H. H., a secretary aged 36, was referred to us because of an unusually severe, patchy, eczematous dermatitis of the feet and hands, with maximal involvement of the feet, which had been present for fifteen months. This was characterized by scaling, erythematous, superficially denuded, crusted, vesicular, fissured plaques on the inner surface of the right foot, extending from the instep to the dorsal surface of the foot and involving part of the ankle, the dorsal surface of the right foot at the base of the first interdigital space and extending into the interdigital space, the instep of the left foot and the medial surface of the left foot just above the heel. The borders of the lesions were not well demarcated; only a few vesicles were seen. In addition, there was a faintly erythematous, rather well demarcated, dry, slightly lichenified plaque on each palm, as well as a few superficial vesicles along the sides of several fingers. All lesions tended to be dry, scaly and somewhat lichenified rather than oozing, although there was some exudation at several sites on the feet.

This condition had had its onset as a rather small, coin-shaped, pruritic, scaling, eczematous plaque on the inner surface of the right foot, approximately fifteen months before the patient consulted us. Soon afterward, there had been progressive extension and increase of pruritus, which was intense and occurred in paroxysms. The course had been characterized by repeated exacerbation and partial remission, but complete clearing had not occurred at any time, and pruritus had not been absent for more than one or two days at a time. The patient had been under the care of a radiologist, who had given her approximately fifteen roentgen ray treatments, during which course of therapy she had had partial improvement and one lesion which had appeared on the flexor surface of the wrist had disappeared

Fig. 5 (case 24).—In some respects case 24 was one of the most interesting in our series despite the apparent insignificance of the involvement. The lesion resembled a fixed drug eruption. It had persisted for almost three years and disappeared within seven days after the responsible food allergens were eliminated. Numerous prompt exacerbations were demonstrated on the addition of food allergens to the diet.

entirely. She had received a total of 1,500 r to all affected sites. Soon after the roentgen ray treatments were discontinued, there had been progressive increase of involvement, and for this reason the radiologist had referred her to us. The management and the course of her eczematous dermatitis during the twelve month period of observation were as follows:

1. Hospitalization was strongly advised, but this was not possible. All previous local medication, which had been quite varied, was discontinued, and 3 per cent

ichthammol in zinc ointment was prescribed. A summary of an allergy work-up, done by Dr. Norman Ingraham Jr., follows:

A. Patch tests of a wide variety of contactants, including eight different constituents of her shoes, and nylons, had entirely negative reactions.

B. Reactions to scratch and intradermal tests with common dust-borne allergens were completely negative.

C. A number of sharply positive reactions to scratch tests with foods were obtained, and it was Dr. Ingraham's opinion that these were of a type which would

Fig. 6 (case 4).—There was an important contributory pyogenic factor in this patient's eruption, and evaluation of the food allergy factor was not possible until the bacterial infection was treated concomitantly.

make one feel that they could be of significance. The foods producing positive reactions were almonds, apples, asparagus, bananas, beets, broccoli, carrots, cashew nuts, cauliflower, American cheese, Roquefort cheese, cherries, cinnamon, coconut, coffee, dates, dill, eggplant, lemons, mushrooms, oats, oranges, peanuts, white potatoes, prunes, spinach, turkey, watercress and wheat. During this two week period there was no restriction of the patient's diet and no improvment.

2. Within one week after she was placed on a scratch test–negative diet, there was 75 per cent improvement in all lesions, with decrease of pruritus. Two weeks

later, there was no additional improvement. Therefore, she was asked to try for one week a basic "nonallergenic" diet consisting only of apricots, peaches, canned pears, prunes, sugar, tea, beans, chicken, string beans, lamb, lettuce, cabbage, peas, ry-crisp,® corn bread, salt, sweet potatoes, turnips, peach preserves and apricot preserves. After one week on this diet, she had complete relief of pruritus and all signs of involvement, except for residual hyperpigmentation, scaling and very slight lichenification, had disappeared.

3. At this time, she went on vacation and disregarded her diet entirely. On successive days, she ate sea food dinners and chocolate candy—indeed, there was no interdiction of foods. Within twelve hours after the first such meal, there was pronounced recurrence of pruritus, followed by a sharp, severe exacerbation involving all previously affected sites. During the next three weeks, she resumed her scratch test–negative diet except that chocolate, nuts and all sea foods were added to the interdicted foods. The pruritus disappeared within three days, and regression of objective signs gradually occurred except for one exacerbation possibly associated with the ingestion of watermelon. At the end of three weeks, the patient was free from pruritus, and only residual hyperpigmentation and slight scaling persisted at the previously involved sites.

4. During the past eight months, this patient noted repeated exacerbations following the attempted addition of the following foods to her diet: wheat bread, or wheat flour in any form, chocolate, shrimp, crab meat, lobster and coffee. In the last three months, she has been able to drink an occasional cup of coffee without exacerbation except for transient pruritus. She has found that complete elimination of wheat products for a period of two or three weeks, as well as interdiction of chocolate, shrimp, crab, lobster and coffee, is followed by complete absence of all signs of involvement. Furthermore, if the ingestion of wheat products is limited to two pieces of part rye and part wheat bread daily, pruritus and scaling of previously affected sites are so minimal that they do not interfere with any of her activities; therefore, she prefers to accept these consequences rather than eliminate bread entirely. Repeated attempts to increase the amount of wheat-containing foods resulted in decided increase of pruritus of the eczematous patches at approximately the original sites of involvement.

The diagnosis was eczematous dermatitis, due to food allergy (wheat, chocolate, coffee, shrimp, crab and lobster), of the hands and feet.

Comment.—The initial elimination diet, which was based on scratch test–negative foods, happened to exclude the principal food allergen, which was wheat, and, also, it resulted in the interdiction of coffee, which was a relatively unimportant food allergen. Complete improvement did not occur on this dietary regimen because of other specific food sensitivities in the form of several different sea foods and chocolate, all of which produced negative reactions in scratch tests. Complete relief of symptoms and disappearance of pruritus followed a brief period of pronounced dietary restriction, which happened to exclude all the responsible food allergens. It is probable that this patient could have been cured by a brief period of hospitalization and the use of a strict trial diet. The rapid response to interdiction of foods would have made it possible to add foods rather rapidly, and within one month she could have been placed on a quite adequate diet.

SUMMARY STUDY AND TREATMENT OF PATIENTS

The successful study and treatment of these 26 patients from the food allergy point of view was accomplished by the following methods: (1) strict trial diet including hospitalization, for 11 patients; (2) basic "nonallergic" diet, for 9 patients; (3) "spot" elimination diet, for 5 patients, and (4) scratch test–negative diet, for 1 patient.

Furthermore, attention to other factors, including careful study and elimination of contactants and primary irritants, elimination of the use of drugs (internal) in several patients, and treatment of the pyogenic factor, was necessary in 12 of the 26 patients. Cure was obtained in only 54 per cent of these 26 patients by determination and elimination of the responsible food allergens alone.

At the same time, it is emphasized that food allergy was such an important contributory factor in the disorders of the other 46 per cent of the patients in whom it was not the only etiologic factor that a satisfactory therapeutic result could not have been accomplished without the discovery and elimination of the allergic foods.

From these data, it would seem that any single dietary method of approach, without consideration of other factors, will result in misconception with regard to the possible primary or contributory food sensitivity factor, and resultant therapeutic failure. In those patients in whom a food sensitivity is the only etiologic factor, prompt improvement and a clinical cure will follow the institution of a suitable elimination diet. We agree with Flood and Perry that the strict trial diet will result in the highest percentage of cures, but the basic nonallergenic diet which we have used on an outpatient basis will be effective for a significant number of patients for whom food allergy is the only etiologic factor. The so-called "spot elimination diet," which simply provides for the interdiction of a small number of foods of high allergenic index, will effect a cure in a fairly small percentage of patients. The failure of any particular dietary regimen tends to discourage the patient and makes it difficult for one to institute a more rigid dietary approach. In general, it is preferable for one to outline a diet which informs the patient what he is to eat, rather than one which tells him what he is not to eat. In the other large group of patients, in whom food sensitivity is only a contributory concomitant factor of varying importance, study and treatment is much less rewarding therapeutically; without hospitalization, it is most difficult for one to untangle and treat the multiple etiologic factors involved.

INCIDENCE OF FOOD ALLERGY IN ETIOLOGY OF ECZEMATOUS DERMATITIS

It is difficult for one to evaluate accurately the role of food allergy in the etiology of eczematous dermatitis so far as the exact incidence is concerned. In the first place, it is not possible for one to exclude food

allergy as an etiologic factor in a given case unless the patient is hospitalized and placed on a strict trial diet and unless there is the elimination of other possible contributory factors, such as pyogenic infection and contactants, which might obscure a concomitant food sensitivity. Obviously, such a procedure is not feasible in a fair percentage of cases. Furthermore, the experience of various investigators will differ considerably. For example, physicians in industry will encounter a higher incidence of eczematous dermatitis due to allergic contactant and primary irritant factors. It has been our experience that food allergy is the only significant etiologic factor for about 7 to 8 per cent of the patients with chronic eczematous dermatitis and an important contributory factor for an additional 8 to 10 per cent of such patients. The common concomitant factors which were recognized in the present series of 26 patients include allergic contactant, primary irritant, pyogenic infection and sensitization and drug allergy.

DESCRIPTION OF LESIONS

We agree with Sulzberger that if food allergens cause eczematous eruptions of the hands "then foods must be considered as possible etiologic agents in other eczematous eruptions no matter what their localization"; and it has been our experience that such eruptions due to specific food sensitivity do occur at other sites on the cutaneous surface.

We have recorded the following sites of involvement in one or more patients: all surfaces of the hands and feet, the eyelids, the forearms, the upper arms, the legs, the thighs, the face, the neck, the groins, the posterior surface of the scrotum, the axillas, the external auditory canals, the trunk and the buttocks. It is true that the hand is the site of predilection, but this is certainly not the only site of localization. In our series of cases, the hands were involved in 21 of 26 patients; the dermatitis was limited to the hands with no other involved sites in only 10 of the 26 patients.

Eczematous dermatitis due to food allergy tends to be patchy rather than diffuse. The dorsal surface and sides of the fingers, the palms, the dorsal surface of the hands and the interdigital spaces occasionally have been sites of localization which we have noted on the hands. On the feet, the patches tend to be localized on the inner and dorsal surfaces of the feet, although the distribution may be on the dorsal surface of the toes, suggesting an allergic contact dermatitis, or on the plantar surface of the feet, suggesting dermatophytosis. Usually the involvement is in the form of plaques, although patients with diffuse involvement of the axillas and groins, as well as with diffuse involvement of the extremities, have been observed. The eyelid lesions are quite

characteristic in that they are fairly well demarcated, erythematous, edematous and slightly scaling and in many respects resemble a fixed drug eruption.

Individual lesions vary considerably. In the vast majority of patients there is a vesicular element, although vesiculation may be absent. There is a tendency to oozing and superficial fissuring, and in many persons vesicopustules and other signs of secondary pyogenic infections are dominant when the patient is first seen. In general, the borders of plaques are not well demarcated, and they are not annular with a tendency to central clearing. Pruritus, which is usually intense, is a regular feature of eczematous dermatitis due to food allergy. The itching is periodic and varies from day to day unless one of the causative foods is eaten daily.

When food diaries are used as a method of study, pruritus is almost as dependable as a sign of an exacerbation as is the appearance of new vesicles and other objective evidence.

However, unfortunately, there are no absolute diagnostic criteria in distinguishing eczematous dermatitis due to food allergy from that due to other factors, but with experience it is possible for one to acquire a "high index of suspicion."

COURSE OF ECZEMATOUS DERMATITIS DUE TO FOOD ALLERGY

In most cases there is a history of a tendency to spontaneous exacerbations and partial remissions. We have not been particularly impressed with a change of incidence in relation to the season of the year, although in general the process tends to be severer during the winter months.

Quite uniformly, rapid, significant improvement has been noted in one to three days when the causative foods were eliminated, provided that the etiologic picture was not complicated by other factors, such as contactants and pyogenic infection. Exacerbations which follow addition of an allergenic food have occurred within fifteen minutes after a small serving of a particular food, and, almost always, objective evidence of such exacerbations is preceded by pruritus. On the other hand, the interval between ingestion and exacerbation has been as long as forty-eight hours after the food was eaten, and in 3 of our patients exacerbations did not occur until the food had been eaten on six consecutive days. It seems quite certain that exacerbations occur more promptly and with a smaller quantity of the allergenic food if the addition to the diet is made within a few days after the initial lesions have become quiescent. Many patients have been observed in whom decided prompt exacerbations occurred after ingestion of a given food soon after initial improvement had occurred on a trial diet; several weeks or months later, these same patients failed to have such exacerbations following the addition of the same food to the diet. It would

seem that in many instances spontaneous desensitization takes place, and several months after disappearance of the patient's dermatitis foods which caused definite exacerbations previously may be taken without resultant symptoms or signs in the skin.

Our experience is at variance with that of Rowe [7b] in that we believe that significant improvement is evident within several days to one week, provided that all the allergenic foods are eliminated from the diet and provided that all other contributory factors have been eliminated and treated. Those patients who have other etiologic factors in addition to food allergy will not improve significantly on a therapeutic approach which is designed to find and eliminate only the food allergy factor.

It is our opinion that the process in many of these patients had its onset as allergic contact dermatitis, and in others as a primary pyogenic dermatitis, for example. Later on in the course of the disease, the specific food sensitivity factor became operative and in many instances became the chief perpetuating factor in the eruption. Also, the reverse was true in that in some instances the eruption had its onset as an eczematous dermatitis due to foods, and subsequently other factors, such as allergic contactants, invasion and sensitization by bacteria, complicated the process. As in allergic contactant dermatitis, it is advantageous for one to treat eczematous dermatitis due to food allergy early in the course of the disease because it is easier to effect a cure under these circumstances. This point was well illustrated in many of our cases.

INCIDENCE OF SENSITIVITY TO VARIOUS FOODS

This series of patients is too small for analysis as to the relative frequency with which individual foods may sensitize the skin, but it is interesting to tabulate the results for what they are worth. In 1 or more patients the allergenic role of the following foods was demonstrated in our series of 26 patients, and in almost all instances exacerbations were noted on 2 or more occasions:

Food	No. of Patients	Food	No. of Patients
Chocolate	9	Grapefruit	5
Sea food *	8	Tomatoes	4
Wheat	6	Eggs	4
Oranges	6	Broccoli	2
Pork	5	Rice	2
Milk	5	Cinnamon	2
Nuts	5	Smoked pork	2

* Lobster, 2; crab, 2; shrimp, 2; clams, 1, and shad, 1.

Specific sensitivity to the following foods was encountered in 1 patient: peaches, carrots, onions, peas, Scotch whisky, butter, veal, pepper, cauliflower, pimento, coffee, pineapple, tea, apricots, cabbage,

string beans, lima beans, chicken, sweet corn, vanilla extract, rye, sweet potatoes, cheese, paprika and white potatoes. At the present time another patient, for whom the primary food allergen is beef, is under observation, as is another, for whom canned pears are the most important food allergen.

COMMENT

It should be emphasized particularly that, while allergic sensitivity to foods is an important reason for chronicity and persistence of an eczematous eruption, it is not an etiologic factor for even a majority of such patients, and study and treatment of the patient in this regard must be carried out with scrupulous regard for the many other possible etiologic factors concerned. In addition, as with many other methods of treatment, too great enthusiasm or "faddism" for diets may have a medically deleterious effect on the whole patient. Prolonged restriction of caloric intake, particularly for underweight patients, for many growing children or for the aged is obviously unjustified. Likewise, the balance of various fractions in the diet and the adequacy of vitamin intake must receive thoughtful consideration. Some patients will cooperate too enthusiastically for their general medical good and may continue to follow severely restricted diets for too long a time and with too little justification.

In general, local treatment was not particularly helpful in the management of this series of cases, except in those in which bacterial infection was an important contributory factor. Also, superficial roentgen ray therapy does not seem to influence significantly the course of eczematous dermatitis due to food allergy. More than 50 per cent of our 26 patients had had an adequate trial of roentgen ray treatment, and, although apparent partial initial improvement had been noted in some patients, the end result was unsatisfactory in all cases in that relapse occurred within a short time after the treatment was discontinued. Penicillin administered parenterally was useful in controlling pyogenic infection when this was a complicating factor.

The history in regard to food sensitivities and other allergic manifestations prior to the onset of the presenting dermatitis was not particularly helpful in the initial evaluation of these patients. There was a family history of allergy for 15 per cent of the patients and a personal allergic (not atopic dermatitis) history for 15 per cent, although the two groups were not identical.

The psychogenic factor seemed to be important for several of our patients, and indeed our experience with the present series leads us to believe that there is an emotional component in the background of the vast majority of persons with food sensitivities. For this reason, one must be careful to evaluate each patient critically and include in the

group of persons with food allergy only those patients who have definite repeated exacerbations following the addition of causative foods to the basic diet. It is conceivable that at least some of these patients could be cured with ideal psychiatric treatment, but this approach has had limited usefulness in our experience. This principle was illustrated in the case of 1 patient who had repeated exacerbations of her dermatitis after the ingestion of small quantities of grapefruit during a period of considerable emotional strain incident to working under an exacting employer; some months later, under more favorable circumstances of employment, it was possible for her to eat several times as much grapefruit with only minimal pruritus and very few objective signs of involvement.

It is essential for one to realize that there is an appreciable individual variation in the course of patients who have eczematous dermatitis entirely or in part due to specific food sensitivity. In some cases a prompt regression of the dermatitis is noted after the interdiction of the responsible foods; in others significant improvement does not occur for as long as seven to ten days. The same variation applies to the onset of an exacerbation following the addition of a causative food, although in the vast majority of cases such exacerbations are seen within a period of forty-eight hours. It is not a simple procedure to manage the patients successfully, and considerable experience is necessary before one acquires an appreciation of the problems involved. The physician who follows a stereotyped approach will not cure a significant percentage of patients with cutaneous food allergy.

SUMMARY AND CONCLUSIONS

1. A series of 26 patients with eczematous dermatitis, for whom it was demonstrated that specific sensitivity to various foods was the only etiologic factor or a significant contributory factor, is presented.

2. The chief localization site encountered was the hands (involved in 81 per cent), although the feet, eyelids, legs, arms, face, scrotum, groins, neck, buttocks, perianal region and axillas were also involved in 1 or more of our patients.

3. In addition to food allergy, other concomitant etiologic factors, including allergic contactants, primary irritants, pyogenic infection and pyogenic sensitization and allergic drug factors, were demonstrated for 12, or 46 per cent, of these 26 patients.

4. Eczematous dermatitis due to food allergy tends to occur in localized, rather inflammatory plaques; oozing, vesiculation and excoriations were noted in a high percentage of cases. Apparent spontaneous exacerbations and partial remissions are relatively common.

5. Pruritis, which was usually quite severe, was a regular and prominent feature of the process.

6. Scratch tests with foods were of almost no value in the management of these patients.

7. The methods of approach in the study of these patients are discussed in some detail. Ideally, the vast majority should be hospitalized for an adequate evaluation from the standpoint of food allergy; for about 40 per cent of our patients such evaluation would not have been possible without hospitalization. However, with a fair percentage of cooperative patients, satisfactory elimination diets can be put into effect on an outpatient basis, especially if food allergy is the only etiologic factor involved.

8. The necessity of evaluating and treating such patients along broad etiologic lines as well as with respect to food allergy is emphasized and illustrated in the case histories of many of our patients.

9. Chocolate, oranges, wheat, pork, eggs, sea food, tomatoes, grapefruit, nuts and milk were encountered more frequently as food allergens in this series of patients, but specific food sensitivity was demonstrated to twenty-six other foods by one or more patients. Sensitivity to two or more foods was demonstrated by 24 of the 26 patients.

10. It is believed that food allergy is one of the important etiologic factors in eczematous dermatitis. In our experience, food allergy is the only etiologic factor or a significant contributory factor in the case of 16 to 18 per cent of patients with chronic eczematous dermatitis of the hands and/or feet.

11. The technic of management and interpretation of results are relatively difficult, especially in the cases of those patients for whom food allergy is only one of numerous possible etiologic factors.

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ABSTRACT OF DISCUSSION

DR. SAMUEL M. PECK, New York: I wish to congratulate Dr. Livingood for the fine piece of work which he did and especially for his patience in carrying out this type of rather ungrateful research. The average dermatologist is somewhat prone to disregard the value of the approach which Dr. Livingood has made because, on the one hand, it is so time consuming and, on the other, the results in many instances are most disappointing. I think the chief difficulty in this problem is the fact that one is unable to judge on clinical evidence alone which cases are due to foods and which are due to the many other factors which can give the same clinical syndrome. Also, it is very rare to have a factor such as food sensitivity play other than a major or a minor role in the dermatitis. Food plays a role in infancy and childhood as the sole cause of eczema. This point no one will deny. However, it is difficult for many to visualize a dermatitis which is limited to the hands as being mainly due to a food factor. Certainly, in the eczema of infancy and childhood, one rarely, if ever, sees limited localization, especially as far as the hands are concerned. It would be a great help if there

were a simple approach to establish or rule out foods as etiologic factors. I have attempted to find one in many instances, but so far, the necessary procedure still boils down to the tedious, long-drawn-out, trial and error type of approach. One thing should be emphasized, namely, that, as I believe, in different parts of the country and probably among different classes of population different combinations of factors are responsible for the dermatitis on the hands. In the few cases in which I believe I have definitely established food allergy as a cause of eruptions on the hands, nearly all the patients were females. If this observation is common, I think it should be helpful. Furthermore, when a patient with a dermatitis on the hands presents himself or herself to me, I automatically lean away from consideration of food as an etiologic factor in the dermatitis of the hands in men; for them fungi, bacteria and contact factors are the predominating etiologic agents. In women, bacteria or molds will also play a role if the eruption is present long enough. I should like to cite 1 case which to me was enough payment in satisfaction and compensated for all the failures with this approach. This was of a young married woman who came to me with a dermatitis of the hands of months' duration. I automatically went through the routine procedures of looking for an infectious agent and for household contacts as the cause of her dermatitis. I totally disregarded the fact that I had treated her successfully for food allergies as the cause of her infantile eczema. In looking over her old records, I found mention of this, and when she eliminated the foods to which she had again become sensitive over the years, the dermatitis on her hands finally cleared up. One must bear in mind that, even when the offending foods are removed, there is no dramatic improvement. Patience and a thorough understanding of all the factors which contribute to the dermatitis are all needed in order for one to treat one of these patients successfully. I wish to congratulate Dr. Livingood and Dr. Pillsbury for an excellent paper.

Dr. Earl D. Osborne, Buffalo: I, too, wish to congratulate Dr. Livingood and Dr. Pillsbury on having cured anyone with a condition having a food factor in an allergic background. It is a very difficult job to undertake. There are a few observations I should like to make. First, I do not believe that it has been proved that protein substances as such will produce eczematous reactions. When one speaks of foods, one must realize that one is talking about a very chemically complex subject. Foods contain not only proteins but also fats, carbohydrates and a multiplicity of oleoresins. I should like to ask Dr. Livingood whether he did any patch testing in his cases with oleoresins present in the various foods. It appears to me that it is possible for one to explain many apparent recurring vesicular eruptions of the hands on the basis of the ingestion of specific oleoresins present in various foods. It is known that in the case of poison ivy and other weed dermatoses a flare-up of a preexisting eruption on any part of the body can be produced by the ingestion of the specific or related oleoresin concerned. I believe that specific hypersensitivity does exist to the oleoresins in many fruits and vegetables, both from local contact and, in recurrences following this, from the ingestion of the specific oleoresin. Finally, it is my belief that investigation will show that when foods are at fault in the production of an eczematous eruption the responsibility is probably that of the oleoresin content and not of the protein, carbohydrate or fat content of the particular food.

Dr. Eugene F. Traub, New York: I, too, enjoyed Dr. Livingood's presentation and wish to thank him. I should like to ask him several questions. Were the cases in which the condition appeared to be influenced by or to react to certain foods discovered through the use of test diets, or did the patients react positively to tests with food to which they were not otherwise known to be sensitive? What

was the percentage of cases in which skin tests with food were helpful? Did any adults in the series that were found to be sensitive to food give any history of having been previously sensitive to that particular food, or had they always enjoyed that food in the past? How long did this newly discovered food sensitivity last? Was it permanent, or did it clear up after the other factors had been removed?

DR. MARION SULZBERGER, New York: I wish to compliment Dr. Livingood and Dr. Pillsbury on an excellent paper. The first point I should like to emphasize is that no one who has not attempted to carry out exact diets on ambulatory patients can realize how difficult the task is. My own results with this method in the last fifteen years have been on the whole very unsatisfactory. I should like to point out that it is almost prohibitively difficult for the patient to carry out a rigid elimination diet. While on such diets the patient can join in almost no social activities—no eating out, no lunches while away from home, no participation in meals with others and no snack or candy or drink between meals, for example. I have found that under these conditions patients either make involuntary slips or keep cheating. The second point I should like to stress is that it is now obvious that atopic dermatitis has an eczematous phase. This is true, not only in infants but in adults, although in the latter it is not as uncommon. This form of atopic eczematous eruption may, of course, be susceptible to flare-ups following exposure to allergenic foods, just as are some cases of infantile eczema. And in adults atopic eczematous eruptions can and do appear on the hands, particularly the dorsa of the fingers.

Did Dr. Livingood exclude the external contact with foods? It is difficult for one to eat bread or oranges, for example, without letting them touch the hands. I think that in order to come to the conclusions that these eruptions are due to ingestion of foods, one must always and absolutely exclude the possibility of external contact, and I feel certain Dr. Livingood has done this.

I enjoyed the presentation and should like to suggest that Dr. Livingood and Dr. Pillsbury may in the future find it advantageous to attempt to classify their cases of "hand eczemas" into two large groups, namely, those of nonatopic and atopic dermatoses.

My impression is that the results following elimination of certain foods would be much greater in atopic persons than in the nonatopic ones. Moreover, for reasons which I mentioned in discussing aberrations of sweating, the role of foods and drugs in exacerbations of dyshidrosis may merit careful scrutiny in the future.

DR. CLARENCE S. LIVINGOOD, Philadelphia: I wish to thank the discussers. Many of the questions brought up in these discussions are answered in our paper. In answer to the questions of Drs. Sulzberger and Osborn, with reference to external contact with food, rather than ingestion, as a factor: We had thought of that point, and it has been our experience that eczematous lesions in some cases are caused by contactant sensitivity to foods, especially citrus fruits and vegetables. However, in our series of cases, external sensitization to foods was a minor factor in comparison with endogenous allergy to foods. We have been careful in evaluating this particular question, and, for example, in some cases suspected foods were given in capsule form.

The duration of sensitivity to a particular food or foods varies in individual cases. In some of our cases the sensitivity disappeared within several weeks after the lesions had disappeared; in others repeated exacerbations could be reproduced as long as six to eight months after the involution of the eruption, and in some it is probable that the sensitivity persists indefinitely. We agree with Dr. Peck that the allergic pattern in regard to food sensitivity in a given case is likely to change over a period of years.

TRAUMATIC MARGINAL ALOPECIA IN WHITE WOMEN

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AND
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LOS ANGELES

COSTA and Junqueira[1] recently summarized the literature on traumatic marginal alopecia, which, according to their observations and those of Ribeiro,[2] occurs almost exclusively in Negro and mulatto women. They pointed out the similarity between this condition and alopecia liminaris frontalis as described by Sabouraud[3] and expressed the opinion that both conditions are caused by stretching the hair in tight braids or "buns" and, especially in the case of Negro women, by efforts to straighten kinky hair. Folliculitis frequently results from this constant traction, and, with or without folliculitis, atrophic changes of the follicles gradually develop and lead to permanent alopecia.

In the cases described by Ribeiro and by Costa and Junqueira the alopecia occurred in triangular areas involving the temples, with the apex pointing downward in front of the ears.

Inasmuch as the previously reported cases of this entity occurred exclusively in Negro or mulatto women, except for 1 case in a white woman, mentioned by Ribeiro, it seemed worth while to record 5 cases in white women.

In view of the fact that the alopecia tends to be permanent if the trauma due to traction from tight hairdress or excessive brushing is allowed to continue, it becomes important for one to recognize the

1. Costa, O. G., and Junqueira, M. de A.: Traumatic Marginal Alopecia Due to Traction on the Hair: A Comparative Study of Alopecia Liminaris Frontalis of Sabouraud, Arch. Dermat. & Syph. **48**:527-532 (Nov.) 1943.

2. Ribeiro, H.: Alopécia marginal traumatica por tracção dos cabellos, Brasil-med. **52**:1267-1271, 1937; Alopécie marginale traumatique, Ann. de dermat. et syph. **9**:495-503, 1938.

3. Sabouraud, R., in Darier, J., and others: Nouvelle pratique dermatologique, Paris, Masson & Cie, 1936, pt. 7, p. 13; De l'alopécie liminaire frontale, Ann. de dermat. et syph. **2**:446-460, 1931; Diagnostic et traitement des affections du cuir chevelu, Paris, Masson & Cie, 1932, p. 398; Pelades et alopécies en aires, ibid., 1929, p. 93.

entity and to advise regarding corrective measures. After atrophy of the follicles has taken place no method of treatment would be likely to succeed. If the causative factors are eliminated early enough, complete recovery takes place, according to the aforementioned observers.

REPORT OF CASES

CASE 1.—D. H., a girl aged 18, came in primarily for the treatment of acne of a moderate degree of activity. In addition to the acne, examination revealed a pronounced thinning of the hair along the sides of the scalp above the ears, where the hair was pulled up toward the top of the head in tight braids. Scattered through the areas of partial alopecia were a number of small follicular papulopustules.

Fig. 1 (case 1).—Area of alopecia in front of and above ear, due to constant traction from tight braids.

CASE 2.—A. C., a girl aged 18, came to the office primarily for the treatment of redness, crusting and slight swelling of the eyelid margins, characteristic of blepharitis. Examination revealed, in addition to the condition of the eyelids, a moderate degree of thinning of the hair on both temple areas in front of the ears. There were a number of broken hairs, some of them with an exclamation point appearance, and a few follicular pustules scattered through these areas. The patient had been in the habit of using metal curlers on the sides of her scalp for the preceding four months. She thought that the thinning of the hair was of about six weeks' duration.

CASE 3.—E. W., a woman aged 65, presented herself with a complaint of an eruption on the sides of the scalp, associated with a thinning of the hair which had been present for about six months. The patient had been using metal curlers at night.

Examination revealed a diffuse thinning of the hair over the scalp, with a mild seborrheal scaling. Involving each temple area in front of the ears there was a more pronounced loss of hair, with a few broken hairs and empty follicles. Just above this area on both sides was a transverse erythematous and slightly elevated ridge or streak which the patient stated coincided with the area covered by the metal curlers. There were a few follicular papules in this erythematous area. A patch test with the patient's aluminum curler gave no reaction.

CASE 4.—D. H., a girl aged 11, had noticed thinning of the hair, with irritation along the temples, for about two weeks. The patient had been in the habit of rolling her hair tightly with metal curlers at night and had been spending a great deal of time every day brushing her hair upward along the sides.

Examination revealed a moderate degree of thinning of the hair along the temples in front of and just above the ears. There were a number of broken

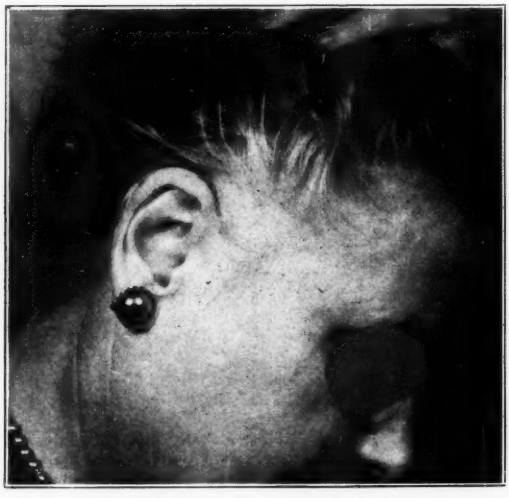

Fig. 2 (case 3).—Triangular area of alopecia involving the temporal region in front of the ear, due to constant traction from the use of tight curlers.

hairs, one of which was shaped like an exclamation point. There was also a moderate degree of redness on each side of the scalp and a number of small superficial papules, several of which were crusted. A patch test was made with nickel, but the patient did not return for observation.

CASE 5.—E. J. S., a woman aged 30, had had a scalp disorder for one and a half years. The patient had been in the habit of combing and brushing the hair upward from the sides and fastening it in a tight braid.

Examination revealed an appreciable thinning of the hair, with some erythema, over the temporal regions. Some of the empty follicles showed dark plugs, and there was evidence of atrophy throughout the areas.

SUMMARY

Five cases are reported of traumatic marginal alopecia in white women.

The clinical appearance consists of a triangular area of partial alopecia involving the sides of the scalp above or in front of the ears, usually accompanied by folliculitis and atrophy of the follicles.

The condition is believed to be caused by persistent traction on the hair from vigorous and prolonged brushing, a tight hairdress or the use of curlers. Previously reported cases of this condition have occurred in Negroes or mulattoes.

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SUPERFICIAL MYCOSES OF VETERANS

II. Dermatophytosis and Cutaneous Moniliasis; Correlation of Clinical Manifestations and Etiologic Agent

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IN THE past few years several efforts have been made to correlate the fungus species with the clinical manifestations of the superficial mycoses. Among these studies have been the work of Lewis, Montgomery and Hopper [1] on the clinical manifestations of Trichophyton purpureum and that of Montgomery and Caspar [2] on the clinical manifestations of Tricophyton purpureum, T. gypseum and Candida albicans.

In a previous paper,[3] we reported on a study of 1,000 veterans with in-service diagnoses of superficial mycoses, 466 of whom had fungous infections as proved by laboratory examination. Of these, 416, or 87.1 per cent, showed dermatophytosis, and 13 (3 per cent), moniliasis. The 416 patients with dermatophytosis and 13 with cutaneous moniliasis had 819 clinical manifestations of various types of dermatophytosis, falling into the commonly recognized categories of tinea pedis, tinea unguium, tinea cruris, tinea manus and generalized tinea, in that order of incidence.

Slides and cultures were made in each case in an attempt to correlate the various types of lesions with the etiologic agents. In general this correlation was most successful with tinea pedis and with tinea unguium. In tinea manus, in contrast, only slight correlation could be

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1. Lewis, G. M.; Montgomery, R. M., and Hopper, M. E.: Cutaneous Manifestations of Trichophyton Purpureum (Bang), Arch. Dermat. & Syph. **37**:823 (May) 1938.

2. Montgomery, R. M., and Caspar, E.: Cutaneous Manifestations of the Fungi Causing Dermatophytosis and Onychomycosis, J. A. M. A. **128**:77 (May 12) 1945.

3. Bumgarner, F. E., and Burke, R. C.: Superficial Mycoses of Veterans: I. Survey of 1,000 Veterans with Service Diagnosis of Dermatophytosis, Arch. Dermat. & Syph. **60**:742 (Nov.) 1949.

made, while in tinea cruris, unfortunately, no dependable correlation could be found. The results of study will be discussed for each category, in the following order: tinea pedis, tinea unguium, tinea cruris, tinea manus and generalized tinea.

TINEA PEDIS

Of the 360 positive cases of tinea pedis, a fungus was isolated in 186, or 51 per cent. T. gypseum was cultured in 118 cases (63 per cent), Trichophyton rubrum in 58 cases (31 per cent), Epidermophyton floccosum in 4 cases (2 per cent) and C. albicans in 6 cases (3 per cent). In other words, T. gypseum was encountered about twice as frequently as was T. rubrum, and both E. floccosum and C. albicans were rarely found. If only the three dermatophytes are considered, T. gypseum was isolated in 64 per cent of the cases, T. rubrum in 34 per cent and E. floccosum in 2 per cent.

The latter figures agree favorably with those of the results obtained by Miss Rhoda Benham, as detailed by Hopkins and associates,[5] who credited her with 111 positive cultures for the periods of 1940 and 1946. The incidence of T. gypseum obtained by Benham averaged 64 per cent; that of T. rubrum, 25 per cent, and that of E. floccosum, 5 per cent. Percentages obtained by Lewis and Hopper[6] in 1938 for 153 cultures were not dissimilar—T. gypseum, 73 per cent; T. rubrum, 25 per cent, and E. floccosum, 2 per cent.

Six types of lesions were identified in cases of tinea pedis: chronic intertriginous, chronic hyperkeratotic scaling, chronic papulovesiculosquamous, bullous, subacute and severe chronic hyperkeratotic scaling. Table 1 details the total number of cases of each of these types, the percentage in which cultures were obtained and the percentage of each of the etiologic agents.

Chronic intertriginous lesions accounted for 41.7 per cent of the 360 clinical cases noted. Cultures were obtained from 48 per cent of the cases. In this type of lesion it is almost impossible to determine the etiologic agent by clinical examination alone. T. gypseum was isolated in 54.3 per cent; T. rubrum, in 36 per cent, and E. floccosum and C. albicans, in 1.4 per cent and 8.3 per cent, respectively.

Chronic hyperkeratotic scaling lesions of the plantar and medial surfaces ranked second, comprising 19 per cent of the 360 cases. Cultures were obtained in only 37.1 per cent of the cases, the majority

4. Footnote deleted on proof.

5. Hopkins, J. C.; Hillegas, A. B.; Ledin, B. L.; Rebell, G. C., and Camp, E.: Dermatophytosis at an Army Post, J. Invest. Dermat. **8**:291 (June) 1947.

6. Lewis, G. W., and Hopper, M. E.: An Introduction to Medical Mycology. Chicago, Year Book Publishers, Inc., 1939.

(84.6 per cent) of the cultures yielding T. rubrum. Three of the 4 cases of chronic hyperkeratotic scaling yielding T. gypseum occurred in Negroes. Several cases of palmar chronic hyperkeratotic scaling due to T. gypseum, in both Negro and white patients, were also noted.

The chronic papulovesiculosquamous type of lesion occurred in 18 per cent, or almost as frequently as did the chronic hyperkeratotic scaling type. It was possible to make cultures from 54.4 per cent of the 68 positive cases. Of these cultures, T. gypseum was isolated in 75.7 per cent and T. rubrum in the others.

Bullous lesions ranked fourth (15 per cent) in incidence. Cultures were isolated in 74 per cent of the cases, T. gypseum being identified in

TABLE 1.—*Type of Lesion and Etiologic Agent in the Cases of Tinea Pedis*

	Chronic Interdigitinous Lesions	Bullous Lesions	Chronic Papulovesicular Squamous Lesions	Subacute Lesions	Severe Chronic Hyperkeratotic Scaling Lesions	Chronic Hyperkeratotic Scaling Lesions	Total
Cases	150	54	68	14	4	70	360
Percentage of total	41.7	15	18.9	4.9	.1	19.4	100
No. cultured	72	40	37	8	3	26	186
Percentage cultured	48	74	54.4	57.1	75	37.1	51
No. yielding T. gypseum	39	40	28	7	..	4	118
Percentage yielding T. gypseum	54.3	100	75.7	87.5	..	15.4	63
No. yielding T. rubrum	26	..	9	1	..	22	58
Percentage yielding T. rubrum	36	..	24.3	12.5	..	84.6	31
No. yielding E. floccosum	1	..	..	..	3	..	4
Percentage yielding E. floccosum	1.4	..	..	..	100	..	2
No. yielding C. albicans	6	..	..	..	..	..	6
Percentage yielding C. albicans	8.3	..	..	..	..	..	3

each instance. Combined infection with T. rubrum was recorded in 2 instances.

Only 14 (5 per cent) subacute lesions (mycosis plus secondary infection) were proved to be tineal. Antibacterial treatment was administered to some patients before positive cultures were obtained. Cultures were obtained in 57.1 per cent of the cases, T. gypseum being identified in 87.5 per cent. Subacute lesions were of vesicular or bullous origin.

Severe chronic hyperkeratotic scaling was noted in only 4 cases (0.1 per cent), from 3 of which cultures yielded E. floccosum. It was necessary to reculture material from 2 of these cases before the organism was obtained.

One may reason that it is possible to identify the etiologic agent by clinical examination alone in the case of bullous and severe chronic hyperkeratotic scaling lesions—that is, T. gypseum in the first instance

and E. floccosum in the second. Of the chronic papulovesiculosquamous lesions, T. gypseum was the etiologic agent in three fourths and T. rubrum in the others. Four fifths of the subacute lesions may be assumed to be caused by T. gypseum. It is not possible to identify by clinical examination alone the fungous species in chronic intertriginous lesions.

In addition to the foregoing conclusions, one may infer from the material in table 1 that T. rubrum is more difficult to isolate than is T. gypseum. In the five types of tinea pedis due to species of Trichophyton, the greater the percentage of lesions from which the organism was isolated, the higher the incidence of T. gypseum noted. For instance, cultures from bullous lesions yielded the etiologic agent in 74 per cent of lesions, T. gypseum being isolated in each case, while cultures from chronic hyperkeratotic scaling lesions yielded the agents in only 37.1 per cent of all lesions, T. gypseum being noted in 15.4 per cent and T. rubrum in 84.6 per cent. One may infer that the incidence of T. rubrum would be much higher in the case of chronic hyperkeratotic, chronic papulovesiculosquamous and chronic intertriginous scaling, and even subacute lesions, if cultures were obtained from all cases listed.

TINEA UNGUIUM

Among the 429 cases of dermatophytosis, there were 245 cases of tinea unguium, of which 222 cases were of infected toe nails and 23 of infected finger nails. Infection of the toe nails was noted and proved by laboratory examination in 60 per cent of the cases of tinea pedis. Hopkins and associates [5] had observed infected toe nails in 47 per cent of their cases of recurrent tinea pedis.

At first, the number of nails involved and the degree of involvement were believed to be of primary importance, but it soon became apparent that there was some correlation between the organism isolated and certain types of involvement of the nail. Therefore considerable effort was expended in an attempt to obtain a reliable key which could be used by physicians interested in this problem. It is not felt that this key is a complete answer, but it is believed that it forms a basis for future and more exhaustive study.

As in cases of tinea pedis, six types of lesions have been noted in cases of tinea unguium; the last two types have been observed only on the hands.

Type 1.—Leukonychia trichophytica, consisting of white patches on the surface or within the nail plate. T. gypseum has been reported as the agent always isolated; however, the mycology laboratory isolated T. rubrum in 3 out of 5 cases in which cultures were obtained.

TYPE 2.—Inverted V infection, with pronounced symmetry, little if any epithelial debris and dulness of the nails (particularly if hyperhidrosis is associated). Usually all the nails on one or both feet are involved. T. gypseum is isolated in the majority of cases.

TYPE 3.—Asymmetric infection, with epithelial debris, thickened nail plate, infected nail bed and involvement of from 1 to 10 nails. If the nails are in the same stage of involvement and, if the nails, particularly those on the second to fifth digits, are dull, T. gypseum is the organism most frequently isolated. If the nails are in varying stages of involvement and retain some luster, T. rubrum is the fungus most often isolated.

TYPE 4.—Ragged, opaque, thick and crumbly nails. Both T. rubrum (in the majority of the cases) and T. gypseum have been isolated from such nails. Often various saprophytes are also present. In 1 instance, mycelium and conidia of Hormodendrum were noted throughout the nail tissue. Several plates were planted with scrapings from the nail, and T. gypseum and Hormodendrum sp. were both isolated.

TYPE 5.—Nail uninfected, separated from infected nail bed. T. gypseum was isolated in 1 case, while in 5 other cases the agent was not obtained. This type of lesion was noted only on the hands.

TYPE 6.—Transverse ridges in nails which are frequently dull or opaque, with a serous exudate at times and occasional paronychia. This is the well known C. albicans type of lesion.

It was possible to culture the organism from toe nails in 33 per cent and from the finger nails in 56 per cent of the cases. The figure given for the toe nails is lower than that obtained in similar studies by Lewis and Hopper, as communicated to one of us (R. C. B.) Final results of the study disclosed that of the 429 patients having dermatophytosis or cutaneous moniliasis, 245, or 57 per cent, had some degree of involvement of the nails. All these, with the exception of 18, showed concurrent involvement of the feet or other parts of the body.

Kittredge[7] stated that generalized involvement of all the nails was unusual and that the case he reported at the time was the seventh one to be reported. Such occurrences, however, are not as unusual as the paucity of literature on the subject would indicate. Three such cases, in all of which T. rubrum was yielded, were uncovered among the veterans included in this study. In addition, 11 cases of tinea unguium of all the toe nails due to T. rubrum were recorded. In 7 of these 11 cases the patient had acquired the infection in Alabama, Mississippi or Louisiana and presented not only type 3 involvement of all the nails

7. Kittredge, H. E.: Onychomycosis Universalis Trichophytina et Epidermophyta: Report of Seventh Case Thus Far Recorded in English, Arch. Dermat. & Syph. **34**:398 (Sept.) 1936.

but also concurrent chronic hyperkeratotic scaling of the plantar surfaces. The strains of T. rubrum isolated in these cases are being studied because of certain morphologic variations.

TINEA CRURIS

Tinea cruris ranked third in prevalence, comprising 86 of the 429 cases of dermatophytosis and cutaneous moniliasis. In only 18 of the 86 verified cases was the infection limited to a single site. In some of these 18 cases there was a history of possible tinea pedis. In 22 cases there were two lesion areas, 18 of which were of the feet and groin. In the remaining 46 cases there were three or more lesion sites.

Three types of lesions were noted: "eczema marginatum" type, a solid plaque type and the erythematous, exudative monilial type. Correlation of the clinical aspects with the etiologic agent has not been possible in the case of tinea cruris. Cultures were obtained in only 33.7 per cent (29 cases) of the verified cases. Of this number, cultures yielded T. gypseum in 8 (27.5 per cent), T. rubrum in 16 (55.4 per cent), E. floccosum in 1 (3.3 per cent) and C. albicans in 4 (13.8 per cent). This small number does not provide sufficiently valid percentages, but comparison of the figures for T. gypseum and T. rubrum (omitting those for C. albicans) indicates that, where the true dermatophytes are concerned, T. rubrum is the etiologic agent in about 66 per cent of the cases of tinea cruris.

TINEA MANUS

Tinea manus ranked fourth in incidence of the clinical types of dermatophytosis. In all, 254 cases of service-diagnosed "ringworm of the hands" were studied. Only 48 cases were established definitely as of tinea manus. The majority of the cases in which this disease was not established were characterized by vesicular and exudative lesions, located particularly on the fingers.

In only 10 of the positive cases were the hands alone involved; in 9 cases the feet and hands were involved, and the remaining 27 yielded three or more positive lesion sites. There was a definite racial distinction observed in that only 5 Negroes (a ratio of 1 Negro to 3 white patients) were shown to have tinea manus. T. gypseum was recovered from lesions on the hands of 2 Negroes and T. rubrum from another.

With regard to origin of infection of the hand and wrist, an interesting factor was noted: One third of the patients with verified lesions had a history of having worked with motor fuels and machinery at the time of onset.

Three definite and distinct types of lesions were observed in tinea manus: a chronic hyperkeratotic scaling, most frequently involving the palms and ventral surfaces of the fingers; a chronic papulovesiculosquamous eruption, which may involve the palm but which most frequently manifests itself on the dorsum of the hand and on the wrist, and a bullous type involving the palmar areas.

The chronic hyperkeratotic scaling type has been observed most frequently; it was seen in 34 (65.4 per cent) of the total of 52 cases of different types of lesions. Scaling is variable but usually fine and branny. It was expected that T. rubrum would always be isolated from this type, particularly since that was the general rule with chronic hyperkeratotic lesions of the feet. However, of the 19 cases in which cultures were obtained only 14 (73.7 per cent) yielded T. rubrum, the cultures in the other 5 (2 Negroes and 3 white patients) having yielded T. gypseum (table 2).

TABLE 2.—*Type of Lesion and Etiologic Agent in Cases of Tinea Manus*

	Chronic Hyperkeratotic Scaling Lesions	Chronic Papulovesiculosquamous Lesions	Bullous Lesions	Not Listed	Total
Cases	34	12	1	5	52*
Percentage of total	65.4	23.8	1.9	9.6	100
Number cultured	19	9	1	5	32
Percentage cultured	55.9	75.0	100	60.0	61.5
Trichophyton gypseum	5	4	1	1	11
Percentage of T. gypseum	26.3	44.4	100	33.3	34.3
Trichophyton rubrum	14	5	0	2	21
Percentage of T. rubrum	73.7	55.5	0	66.6	65.7

* Four cases with both chronic hyperkeratotic scaling and chronic papulovesicular squamous lesions were observed.

Chronic papulovesicular squamous lesions of the dorsum of the hand and wrist were observed in 12 cases. In 9 of these, cultures were obtained: In 4 the organism was identified as T. gypseum and in 5 as T. rubrum. Several patients with this type of lesion of the hands had generalized dermatophytosis as well. The bullous type of tinea manus was observed only once, on the thenar and hypothenar eminences of the left hand of a 26 year old Negro. He also had bullous lesions of the feet. T. gypseum was identified from cultures of both areas.

Of the 32 cultures made in cases of tinea manus, in 21, or 65.7 per cent, T. rubrum was identified. We believe that the greater part of the 44 per cent of uncultured chronic hyperkeratotic scaling lesions and of the 25 per cent of uncultured chronic papulovesicular squamous lesions were probably due to T. rubrum. This belief rests, in part, on evidence presented in table 1 and table 2, which indicates that T. gypseum is

easier to isolate than is T. rubrum. Additional evidence rests on correlation of types of lesions with etiologic agents. In other words, if a patient presents a chronic hyperkeratotic lesion of the feet from which T. rubrum has been isolated and a chronic hyperkeratotic lesion of the hand not yielding a culture, one may expect with a high degree of certainty that T. rubrum is also the etiologic agent of the tinea manus.

TABLE 3.—*Dermatophytosis and Cutaneous Moniliasis*

	Number of Patients	Total Number of Patients	Percentage
Patients with single sites of infection		183	42.6
Feet	130		
Toe nails	18		
Groin	18		
Hand	10		
Finger nails	1		
Buttocks	4		
Leg	1		
Trunk	1		
Patients with two sites of infection		179	41.7
Feet, nails	140		
Feet, groin	18		
Feet, hands	9		
Feet, finger nails	2		
Groin, finger nails	2		
Groin, perineum	1		
Groin, toe nails	1		
Toe nails, finger nails	6		
Patients with three sites of infection		33	7.7
Feet, nails, groin	18		
Feet, nails, hand	7		
Feet, nails, finger nails	1		
Feet, hand, leg	1		
Feet, groin, perineum	1		
Feet, groin, upper trunk	1		
Hand, groin, neck	1		
Nails, groin, buttocks	3		
Patients with four sites of infection		21	5.0
Feet, nails, hand, finger nails	6		
Feet, nails, groin, axilla	2		
Feet, nails, groin, hand	2		
Feet, nails, hand, leg	4		
Feet, nails, leg, groin	1		
Feet, nails, abdomen, groin	1		
Feet, nails, groin, buttocks	4		
Groin, leg, arm, axilla	1		
Patients with generalized infection		13	3.0
Total		429	100

GENERALIZED DERMATOPHYTOSIS

Table 3 lists patients with verified dermatophytoses according to sites: For 42.6 per cent (183 patients) there was evidence of one positive site; for 41.7 per cent (179 patients), of two positive sites; for 7.7 per cent (33 patients), of three positive sites; for 5 per cent (21 patients), of four positive sites, and in 3 per cent (13 patients), of five or more positive sites. For the last-mentioned patients the infection was termed generalized; 10 of the 13 patients were white, and 3 were

Negroes. Since white persons constituted two thirds of the total number studied, incidence by ratio was about the same for white persons as for Negroes.

Cultures were isolated from 10 of the patients: T. rubrum was obtained from 8 white patients and T. gypseum from 1 Negro and 1 white person. Since T. rubrum is more difficult to isolate than is T. gypseum, one may assume that T. rubrum is the cause of more than 80 per cent of the cases of generalized dermatophytosis.

The most extensive case observed by us was that of R. T., a 28 year old machinist, who stated that he had incurred the infection four years previously while he had been in the Navy. The condition had remained chronic since that time and had gradually spread to cover three fourths of the body. Both hands and feet showed chronic hyperkeratotic scaling of the palmer and plantar surfaces. All toe nails and finger nails were infected. Lesions of the chronic papulovesiculosquamous type extended from the area of the groin over the abdomen and buttocks and down the inner aspects of the thighs almost to the knees. Similar lesions covered the forearms, the shoulders, the upper portion of the trunk and the neck and even extended onto the scalp. T. rubrum was isolated from several areas.

TABLE 4.—*Sites of Generalized Tineal Infections*

Total Number of Patients	13
Total Number of Sites	95
Average Number of Sites per Patient	7.3

Site	Number of Patients	Site	Number of Patients
Feet	12	Finger nails	5
Groin	11	Abdomen	4
Buttocks	9	Scalp	4
Hands	9	Axilla	4
Toe nails	8	Concha of ear	4
Upper part of trunk	8	Arm	3
Legs	7	Face	1
Neck	6		

SUMMARY

1. Of the various clinical types of dermatophytosis, tinea pedis ranked first in incidence, followed in order by tinea unguium, tinea cruris, tinea manus and generalized tinea.

2. Multiple clinical types of dermatophytosis were noted in more than half the patients with verified infections.

3. Chronic hyperkeratotic scaling is recognized as being the most frequent type of tinea manus, occurring in about 66 per cent of the verified cases.

4. Infected toe nails occurred in 60 per cent of the cases of tinea pedis. In only 18 of the 245 cases of tinea unguium was there no clinical or laboratory evidence of concurrent dermatophytosis.

5. T. gypseum was identified as the etiologic agent in about two thirds of the cases of tinea pedis yielding cultures and in about 60 per cent of the cases of tinea unguium yielding cultures. T. rubrum was identified as the etiologic agent in two thirds of the cases of tinea cruris and tinea manus.

6. Correlation of types of lesion with species of fungus has been most successful in cases of tinea pedis and tinea unguium. Six types of lesions were noted in each.

7. The search for multiple clinical types should be painstaking, since the detection of the type aids not only in ultimate eradication but also in the clinical identification of the etiologic agent.

8. Statistics indicate that it is the etiologic agent rather than the type of lesion which influences the possibility of isolation. T. gypseum is easier to isolate than is T. rubrum.

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SARCOMA INVOLVING THE SKIN

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TRADITIONALLY tumors have been divided into those of ectodermal and those of mesodermal origin. The malignant neoplasms have been similarly divided into carcinoma, including melanoma, and sarcoma. However, this classification is not completely satisfactory, since in some cases epithelioma, especially melanoma, reacts in the body in the same highly malignant manner as sarcoma. Furthermore, in some cases sarcoma—in many of the cases neurosarcoma—is actually of ectodermal origin. However, common usage makes this classification the most workable at this time.

A voluminous literature has developed around the problems presented by cases of cutaneous epithelioma and melanoma. While sarcoma is not completely neglected, comparatively little can be found in dermatologic writings concerning it. Beerman,[1] in his excellent review on tumors of the skin, did not find enough to enable him to consider sarcoma. Yet the dermatologist is often called on to diagnose and eradicate the smaller sarcomatous lesions. As will be demonstrated later, sarcoma is among the most difficult to diagnose in the field of cutaneous neoplasms. Furthermore, as in the case of epithelioma, cure depends on early recognition of the growth and prompt, adequate therapy.

MATERIAL

The statements made in this paper are based on a study of 28 patients with histologically proved sarcoma involving the skin. Six of the patients were encountered in private practice. The remainder were studied from records made available by cooperating physicians and hospitals.[2] In this series all of the neoplasms except 1, diagnosed as rhabdomyosarcoma, are believed to have originated in the skin and its appendages. Cases of special types of sarcoma, well recognized by

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1. Beerman, H.: Tumors of the Skin: A Review of the Literature, Am. J. M. Sc. **211**:480 (April) 1946; **212**:479 (Oct.) 1946.

2. This includes Highland-Alameda County Hospital, Mount Zion Hospital, Oakland Veterans Administration Hospital, Drs. Norman N. Epstein, Herman V. Allington, J. O. Smith, Franklin I. Harris, John Sampson and Allan Cohen.

TABLE 1.—*Cases of Cutaneous Sarcoma*

No.	Sex	Age, Yr.	Duration	Race	Location	Metastases	Histology	Prognosis	Treatment
1	M	57	6 mo.	W	Shoulder	+	Giant cell sarcoma	Died 3½ yr.	Excision, amputation, roentgen irradiation, cyclotron irradiation
2	M	44	6 mo.	W	Occiput	0	Fibrosarcoma	Living and well 2 yr.	Excision
3	M	23	3 mo.	W	Postauricular area	0	Fibrosarcoma	Living and well 7 yr.	Excision
4	M	55	9 mo.	W	Forehead	0	Fibrosarcoma	?	Excision
5	F	52	1 yr.	N	Inguinal region	+	Mixed cell sarcoma	Died 1 yr.	Excision, roentgen irradiation
6	M	15	6 mo.	W	Scapular	0	Fibrosarcoma	Living and well 3 yr.	Excision
7	M	70	4 mo.	W	Thigh	+	Giant cell sarcoma	Died 1 yr.	Excision
8	F	48	6 yr.	W	Thigh	+	Fibrosarcoma	Died 6½ yr.	Excision
9	M	46	5 mo.	W	Thigh	+	Fibrosarcoma	Died 1 yr.	None
10	M	45	4 yr.	W	Arms, legs	0	Neurosarcoma (von Recklinghausen's disease) *	Died 10 yr.*	None
11	F	26	1 yr.	W	Thigh	0	Fibrosarcoma	Living and well 8 yr.	Excision
12	M	26	1 yr.	N	Shoulder	+	Fibrosarcoma	Died 1½ yr.	Excision, roentgen irradiation (12,700r)
13	M	77	?	W	Preauricular area	0	Fibrosarcoma	Died 2 yr.*	Excision
14	F	47	1 yr.	W	Thigh	+	Rhabdomyosarcoma	Died 2½ yr.	Disarticulation
15	F	28	4 yr.	W	Popliteal space	+	Fibrosarcoma	Died 4 yr.	Excision
16	M	62	2 yr.	N	Arm	+	Fibrosarcoma	Died 2 yr.	None
17	M	56	15 mo.	W	Scapular	0	Fibrosarcoma	Living and well 2 yr.	Excision
18	F	49	5 yr.	W	Thigh	0	Neurosarcoma	Living and well 6½ yr.	Excision
19	F	32	3 mo.	W	Abdomen	0	Angiosarcoma	Living and well 9 mo.	Excision
20	M	63	20 yr.	W	Groin	+	Myxosarcoma	Died 22 yr.	Excision, roentgen irradiation
21	M	39	1 yr.	W	Foot	+	Fibrosarcoma	Died 2 yr.	Excision
22	M	43	19 yr.	W	Buttock	+	Fibrosarcoma	Died 20 yr.	Excision, roentgen irradiation, phosphorus^{32} irradiation
23	M	67	2 yr.	W	Back	+	Fibrosarcoma	Living, with recurrence 7 yr.	Excision, roentgen irradiation
24	F	42	3 mo.	W	Thigh	0	Fibrosarcoma	Living and well 10 yr.	Amputation
25	F	8	2 yr.	W	Face	+	Angiosarcoma	Died 3 yr.	Excision, roentgen irradiation
26	M	1½	1 yr.	W	Axilla	0	Angiosarcoma	?	Radium therapy
27	F	80	1 mo.	W	Lip	0	Fibrosarcoma	?	Curettage and desiccation
28	F	37	4 mo.	W	Arm, thigh	0	Fibrosarcoma	?	Curettage and desiccation

* The patient died of causes other than sarcoma.

dermatologists, as Kaposi's sarcoma and lymphosarcoma, were not included in this study. Table 1 presents the 28 cases included in the series.

CLINICAL PICTURE

Sarcoma develops in the skin or the subcutaneous tissue as a small nodule. If superficially located, it grossly resembles epithelioma. Growth may be rapid or slow, although the former is more common. The nodule develops to form a mass which becomes elevated, discolored (usually purplish) and later necrotic and ulcerated. Bleeding is common. Later, the necrotic ulcer becomes fungating and granulomatous. Involvement of regional lymph nodes is comparatively infrequent, this having been noted clinically in only 2 cases (7.1 per cent).

Only a single tumor was present in 24 cases (86 per cent). Multiple tumors were found in the other 4. Table 2 gives the location of these tumors. There were 61 lesions in the 28 patients. The most important finding is that in 39.4 per cent of the cases the lower extremities were

TABLE 2.—*Location of Tumors in this Series*

Location	Cases
Head	6
Trunk	10
Upper extremities	3
Lower extremities	11

involved. In 35.8 per cent the trunk was affected. Sarcoma was noted on the thighs in 28.6 per cent. Epithelioma is definitely unusual in these locations.

CLINICAL DIAGNOSIS

Sarcoma is easily diagnosed from its clinical appearance when it has become a massive purplish tumor with necrosis, ulceration and fungating overgrowth. However, in no instance in this series was the clinical diagnosis suggested on simple inspection of a small growth. The clinical diagnoses in 18 cases in which a tentative diagnosis was offered prior to surgical intervention are given in table 3. In the 3 instances in which the tumor was correctly diagnosed, the lesion was far advanced. In the personally observed 6 cases the diagnosis was sarcoma in 1 (a large necrotic mass), squamous cell epithelioma in 1, basal cell epithelioma in 2, granuloma pyogenicum in 1 and fibroma in 1. The only other common diagnosis in the other 22 cases was infectious granuloma, including cutaneous tuberculosis, in 3 cases. Therefore, the most important differential problem is that of epithelioma. The coincident, adjacent occurrence of epithelioma and sarcoma in 2 cases further complicated the problem of diagnosis.

Table 4 outlines the differential points between sarcoma and epithelioma. The most important features may be briefly mentioned. Of the cases of sarcoma, 35.8 per cent occur in the precancer age. The most common locations for sarcoma are on the extremities and the trunk, areas comparatively infrequently involved by epithelioma. The massive growth and necrosis usually make the differentiation obvious. Sarcoma is more frequent in Negroes than is epithelioma, and this may be of importance in some cases. Sarcoma usually grows more rapidly than epithelioma. The lesions of both sarcoma and epithelioma are usually single but may be multiple in either case. Sarcoma often develops in

TABLE 3.—*Preoperative Diagnosis in 18 Cases of Sarcoma Involving the Skin*

Diagnosis	Cases	Diagnosis	Cases
Epithelioma	4	Hernia	1
Squamous cell	2	Gout	1
Basal cell	2	Polyarthritis	1
Sarcoma	3	Draining sinus tract	1
"Infectious granuloma"	2	Fibroma	1
Infected wound	1	Granuloma pyogenicum	1
Sebaceous cyst	1	Cutaneous tuberculosis	1

TABLE 4.—*Differential Diagnosis Between Sarcoma and Epithelioma*

Differential Point	Sarcoma	Epithelioma
Age	35 per cent less than 40	Usually over 40
Location	Trunk and lower extremities	Face and dorsa of hands
Clinical picture	Massive overgrowth	Ulceration
Negroes	More frequent	Less frequent
Growth	More rapid	Slower
Onset	Cutaneous or subcutaneous	Cutaneous
Surgical findings (gross)	Necrosis, greater spread than expected	Nodular and ulcerative
Metastases	Common	Comparatively infrequent
Lymph node involvement in presence of metastases	Infrequent	Probably always

the subcutaneous tissue, although it commonly appears in the true skin originally.

While simple inspection and palpation may not establish the correct diagnosis, two features are found at surgical intervention that give one invaluable clues to the true nature of the neoplasm. Even in small size, sarcoma extends more deeply than one would expect epithelioma of the same apparent size to do and, second, necrosis is present. These features have been of great importance in this series.

ETIOLOGY AND INCIDENCE

Obviously, the cause of sarcoma is unknown. However, there are certain known factors of interest. For instance, certain precarcinomatous lesions may also be presarcomatous. In this group might be included

lupus vulgaris, xeroderma pigmentosum and radiodermatitis. MacKee [3] and others have stated that it is doubtful that sarcoma ever develops in areas of radiodermatitis. While cases have been reported in which a lesion developing in such an area histologically resembled sarcoma, he felt that it was actually of epithelial origin. In case 2 of this series a lesion diagnosed as sarcoma developed less than 1 inch (2.5 cm.) from a lesion diagnosed as squamous cell epithelioma in an area of radiodermatitis. The former was of greater duration than the latter, but both lesions were present at the same time and were excised in a single block dissection. Histologically there was no resemblance between the two lesions. After a study of special stains (Masson's, silver impregnation and phosphotungstic acid) Biskind [4] felt that the sarcomatous lesion was of mesodermal origin.[4a] In case 13 three lesions were removed from the face of a 77 year old man at the same time. Histopathologic examination revealed that one lesion was basal cell epithelioma, one was mixed cell epithelioma and the third sarcoma.

Six apparently unrelated conditions occurred concurrently with sarcoma in this series. Four—psoriasis, lupus erythematosus, polyarthritis and cholelithiasis—were obviously coincidental, and each occurred in 1 case only. Tuberculosis was noted twice. Sarcoma was associated with carcinoma in 3 instances. This is 10.7 per cent of the entire series but is 16.6 per cent of the patients over 40 years of age, i. e., in the cancer age.

The evidence presented in the two previous paragraphs causes one to speculate whether the factor that leads to the development of sarcoma is not the same as that which leads to carcinomatous degeneration. Apparently there is in certain patients and certain lesions a tendency toward malignant changes, and the malignant cells may be of epidermal or mesodermal origin.

Trauma seemed to be important in the development of sarcoma in 5 cases, 17.8 per cent. In 4 cases the patient related the onset of the tumor to a severe traumatic incident involving the affected area. These included a kick in the buttocks in a fight, a home accident, an automobile accident and an industrial accident. In the fifth case, apparently a recurrent low grade sarcoma of twenty years' duration became actively malignant, the growth was accelerated and death occurred two years after an automobile accident. The literature is filled with arguments

3. MacKee, G. M.: X-Rays and Radium in the Treatment of Diseases of the Skin, ed. 3, Philadelphia, Lea & Febiger, 1938, p. 355.

4. Biskind, G.: Personal communication to the author.

4a. According to a report received from Major Robert B. Franklin, Medical Corps, United States Air Force, dated July 29, 1949, this patient was hospitalized because of metastases in the left pleura and the liver. Such metastases are much more suggestive of sarcoma than of epithelioma.

regarding the importance or the lack of importance of trauma in initiating malignant changes. Five more cases will not settle these arguments.

There were 17 men and 11 women in this series. However, in other reported studies there does not seem to be any significant difference in sex incidence.

Twenty-five of the patients were white; the other 3 were Negroes. Apparently, the Negro does not possess immunity from sarcoma parelleling his comparative freedom from certain other types of cancer, including basal cell epithelioma. Furthermore, all 3 Negroes died of their sarcoma in an average of eighteen months. The longest survival in a Negro in this group was two years. This is a much shorter period of survival than the average for the entire series (5.35 years).

The age at the time of onset is given in table 5. This demonstrates that no age group is exempt. The youngest patient was 1½ years of age; the oldest, 80 years of age. Thirty-five and seven-tenths per cent of the patients were less than 40 years old—the so-called precancer age.

TABLE 5.—*Age at Onset of Sarcoma*

Years	Cases	Years	Cases
0 to 10	2	41 to 50	8
11 to 20	1	51 to 60	4
21 to 30	4	61 to 70	4
31 to 40	3	71 to 80	2

The 6 personally observed cases were culled from about 9,000 dermatologic cases, an incidence of 1 in 1,500. However, this incidence seems high, as I do not recall seeing sarcoma in any other group of dermatologic patients that have been under my care. According to Eller,[5] primary sarcoma of the skin accounts for only 5 to 6 per cent of cases of all types of sarcoma occurring in the human body.

PATHOLOGY

It is not the purpose of this paper to reiterate the well established criteria for the histologic diagnosis of sarcoma. However, it should be realized that despite clearcut textbook descriptions it is often difficult to establish the diagnosis of sarcoma. For instance, MacKee,[3] as previously stated, claimed that the lesions arising in areas of radiodermatitis that have been diagnosed as sarcoma by general pathologists are in reality spindle cell epithelioma. Montgomery [6] stated that most so-called

5. Eller, J. J.: Tumors of the Skin: Benign and Malignant, 1939, Lea & Febiger, Philadelphia, p. 434.

6. Montgomery, H.: Epitheliomas of the Arm Simulating Endothelioma, Sarcoma and Sporotrichosis: Two Unusual Cases, M. Clin. North America **19**:605 (Sept.) 1935.

cases of sarcoma of the extremities are actually cases of epithelioma. Furthermore, it is often difficult to determine whether a lesion is a sarcoma or very cellular, nonencapsulated benign fibroma. This is well illustrated by the case reported by Eller and Kest.[7] Biopsy specimens were sent to a number of leading skin and tumor pathologists. The following diagnoses were offered by these admitted experts: D. L. Satenstein and D. Wilbert Sachs—"small spindle cell sarcoma with hemorrhage"; Emmerich von Ham—"fibrosarcoma"; Hamilton Montgomery—"angiosarcoma"; Paul Klemperer—"fibrosarcoma"; J. Frank Fraser—"fibrosarcoma"; Fred D. Weidman—"fibrosarcoma"; Francis Carter Wood—"neurofibroma." In a study of cases presented before various dermatologic societies in this country it is obvious that this is not an unusual experience.

This demonstrates the difficulty of accepting histologic observations as the only proof of the diagnosis of sarcoma. It is a medical axiom that the laboratory results must be correlated with the clinical findings in all

TABLE 6.—*Histologic Types of Sarcoma*

Type	Cases
Fibrosarcoma	18
Angiosarcoma	3
Giant cell sarcoma	2
Neurosarcoma	2
Mixed cell sarcoma	1
Rhabdomyosarcoma	1
Myxosarcoma	1

branches of medicine. Therefore, it is suggested that the diagnosis of sarcoma must be based not only on the histopathologic aspects but on the clinical findings and on the course of the neoplasm as well.

In this series the diagnoses were made by 12 different pathologists. While any given histopathologic section was examined by more than one pathologist in only a few instances, the fact that so many pathologists were involved ruled out the possibility that this series was influenced unduly by personal factors on the part of the examiner. Furthermore, the mortality of 54.2 per cent speaks eloquently for the obvious fact that, whatever criteria are employed, most of these cases were instances of highly malignant sarcoma.

The histologic types of the tumors are given in table 6. The cases of giant cell sarcoma may be included in the group of cases of fibrosarcoma, so that the latter diagnosis was made in 71.5 per cent of the total number of cases of sarcoma of the skin. In other words, fibrosarcoma occurred three times as frequently in the skin as did all the other forms of sarcoma

7. Eller, J. J., and Kest, L. H.: Sarcoma Arising in an Organized Hematoma Resulting from a Single Trauma, Arch. Dermat. & Syph. **43**:813 (May) 1941.

combined. This is in contrast to the statements of Stewart and Copeland [8] and of Cutler, Buschke and Cantril,[9] who expressed the belief that soft tissue sarcoma is in most cases neurosarcoma. In only 2 cases of this series was there evidence of associated von Recklinghausen's disease or of a tumor of a nerve trunk or of an unusual amount of nerve tissue in the sarcoma.

Autopsies were performed in 9 cases. Metastatic lesions were demonstrated in all 9 and, furthermore, the cell type of the metastases was identical with that found in the primary cutaneous tumor. Death was due to cachexia or to terminal incidents, such as bronchopneumonia or nephritis. The locations of the metastatic lesions are listed in table 7. This table indicates that metastases occurred most frequently in the lungs and the pleura, followed by the bones, the skin, the lymph nodes and the liver. Autopsies revealed involvement of the deep lymph nodes more

TABLE 7.—*Locations of Metastases Observed in 9 Autopsies*

Location	Cases	Location	Cases
Lungs and pleura	8	Heart	1
Skin	4	Kidney	1
Lymph nodes	4	Spleen	1
Bones	4	Peritoneum	1
Endocrine glands (adrenals, pancreas, pituitary)	3	Omentum	1
Liver	3	Gallbladder	1
		Subcutaneous tissue	1

often than one would expect on clinical examination, suggesting that dissemination may occur through the lymphatic channels as well as through the blood stream.

PROGNOSIS AND TREATMENT

Obviously, therapy is imperative in this highly malignant condition. In 13 of 24 cases in which there was a follow-up period of nine months or more, the patient had already died of metastases at the time of the last observation. This is a mortality rate of 54.2 per cent. Undoubtedly, this percentage would increase with more prolonged observation. Two patients died of other causes, and their deaths are not included as resulting from sarcoma. In these 13 cases the average period from the onset of symptoms to death was 5.4 years. In the 11 cases in which the patient is still living or has died from other causes the average survival is 5.3 years. The total for the entire group was 5.35 years.

8. Stewart, F. W., and Copeland, M. M.: Neurogenic Sarcoma, Am. J. Cancer **15**:1235 (July) 1931.

9. Cutler, M.; Buschke, F., and Cantril, S. T.: Cancer: Its Diagnosis and Treatment, Philadelphia, W. B. Saunders Company, 1938, pp. 659-666.

The longest life was twenty-two years, and the shortest in the fatal cases was one year after the appearance of the primary tumor.

The earlier treatment is instituted, the better the prognosis. If one employs a duration of six months as an arbitrary dividing line between early and delayed treatment, one finds that the mortality in the early-treated group was 30 per cent of the 10 patients receiving prompt attention. On the other hand, the mortality in 14 receiving treatment more than six months after the onset was 71.4 per cent. While the number of cases studied may not be significant, these figures support the obvious thesis that the earlier adequate treatment is instituted, the better the prognosis. It is to the credit of both the patients and the physicians that almost 35 per cent of the patients received treatment less than six months after the appearance of the tumor.

Three patients were untreated throughout the course of their sarcoma. All died, the average period of survival being 4.3 years. However, this

TABLE 8.—*Mortality with Each Type of Treatment*

Type of Treatment	Patients	Mortality
Excision	21	9 (42.7%)
Roentgen irradiation	7	6 (85.8%)
Amputation	3	2 (66.6%)
Curettage and electrodesiccation	2	0
Cyclotron irradiation	1	1 (100%)
Phosphorus ^{32} irradiation	1	1 (100%)
Radium irradiation	1	0
None	3	3 (100%)

comparatively favorable average is unduly influenced by the inclusion of a patient with a questionable diagnosis of low grade neurosarcoma. This patient died ten years after the onset of his sarcoma, but death was due to an intercurrent tumor of the bladder diagnosed as carcinoma. The average for the other 2 patients was 1.5 years, a figure which is probably closer to the true outlook for patients with untreated cutaneous sarcoma.

It is difficult to compare different schemes of treatment because the prognosis depends on many factors, such as duration of the lesion prior to the onset of treatment, race, thoroughness of treatment and presence or absence of metastases. Furthermore, a small series does not allow dogmatic statements. Many patients received more than one form of treatment. Table 8 shows the number of patients receiving each type of treatment and the number dying despite the approach utilized.

It is important to note that more than 50 per cent of the patients treated by excision, with or without auxiliary measures, were still living at the time this study was conducted. Furthermore, of 13 patients treated with excision alone, 7, or 53.8 per cent, survived. Of 6 patients whom I personally treated by radical cautery excision, 4 are living and

well without evidence of recurrence or metastases after an average survival period of 2.75 years. One patient succumbed to metastases three years postoperatively. There was no opportunity to make follow-up studies on the sixth patient.[10]

Amputation does not offer a better prognosis than simple excision, provided, of course, that the latter is adequately performed. In addition, excision avoids the permanent disability that follows amputation. For instance, patient 11 presented a large nodular fibrosarcoma of the thigh of one year's duration. The histologic section was classified as grade 2. The lesion was completely excised, and the patient is living and well eight years later. Of perhaps even greater importance, she has been able to lead a normal life, having had two successful pregnancies since and being able to raise her children.

Irradiation may cause regression of the tumor, but there is no evidence that it is curative. Most authorities agree that these types of sarcoma are radioresistant. Radiation therapy should not be used as a substitute for surgical removal nor can postoperative radiation act as a safety mechanism to compensate for inadequate excision.

SUMMARY

A study of 28 patients with histologically proved sarcoma involving the skin is presented.

The lesions start as small nodules, which become massive, necrotic, ulcerated, fungating tumors.

Epithelioma is the most important of the lesions to be differentiated from sarcoma and the most difficult to differentiate.

Precarcinomatous lesions may be presarcomatous, and patients with sarcoma may have associated carcinoma.

To diagnose sarcoma on purely histologic grounds is difficult and may be fallacious. The histologic observations should be correlated with the clinical findings and course.

The earlier treatment is instituted, the better the prognosis. The mortality was almost 250 per cent greater in the group receiving treatment after six months than in those being cared for less than six months from the onset of the tumor.

Adequate excision is apparently the treatment of choice.

447 Twenty-Ninth Street.

10. These statistics must be modified, as metastastic lesions developed in 1 other patient while this paper was in press. See footnote 4a.

BERYLLIUM GRANULOMAS OF THE SKIN

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CINCINNATI

IN A RECENT paper, Grier, Nash and Freiman [1] have reviewed the literature relating to the effects which beryllium may have on the skin and subcutaneous tissues. They recognized four distinct types of cutaneous reaction to beryllium. Contact dermatitis occurs in persons working with soluble salts of beryllium, particularly beryllium fluoride or beryllium sulfate.[2] Ulcers result when crystals of soluble beryllium salts become embedded in the tissues; these heal only after the crystals have been removed surgically or have been extruded spontaneously.[2] In some patients suffering from chronic pulmonary berylliosis, granulomas of the skin occur, and these are similar to the granulomas which develop in the lungs.[3] Grier and his co-workers have observed a fourth variety of cutaneous reaction to beryllium which they term "subcutaneous granuloma occurring in persons who cut themselves on fluorescent lamps."

Tissues received from 4 persons in whom subcutaneous granulomas developed after beryllium compounds had been traumatically introduced into the skin have been examined at the Kettering Laboratory. We have also received tissues from an additional person in whom granulomatous inflammation of the subcutaneous tissues followed the introduction of metallic beryllium.

CLINICAL DATA

The clinical data were essentially similar for all 5 patients. In 4 the beryllium was embedded in the skin as the result of the accidental breaking of fluorescent lamp tubes and the puncturing of the skin by

This work was done under a contract with the Office of Naval Research.

From the Kettering Laboratory of Applied Physiology, University of Cincinnati, College of Medicine.

1. Grier, R. S.; Nash, P., and Freiman, D. G.: Skin Lesions in Persons Exposed to Beryllium Compounds, J. Indust. Hyg. & Toxicol. **30**:228-237, 1948.

2. Van Ordstrand, H. S.; Hughes, R.; De Nardi, J. M., and Carmody, M. G.: Beryllium Poisoning, J. A. M. A. **129**:1084-1090 (Dec. 15) 1945.

3. Hardy, H. L., and Tabershaw, I. R.: Delayed Chemical Pneumonitis Occurring in Workers Exposed to Beryllium Compounds, J. Indust. Hyg. & Toxicol. **28**:197-211, 1946. Pyre, J., and Oatway, W. H., Jr.: Beryllium Granulomatosis, Alias Miliary Sarcoid, Salem Sarcoid, Miliary Sarcoidosis, Chronic Beryllium Poisoning, or Delayed Chemical Pneumonitis, Arizona Med. **4**:21-29, 1947.

fragments of glass. The fluorescent powder which lines tubes of this type is comprised of a calcined mixture of zinc oxide, manganese oxide, beryllium oxide and silica. In these 4 patients the lacerations healed with production of excess fibrous tissue. Two of the lesions, when first seen by physicians, were believed to be simple keloids. All four lesions, on one or more occasions after initial healing, opened to discharge thick,

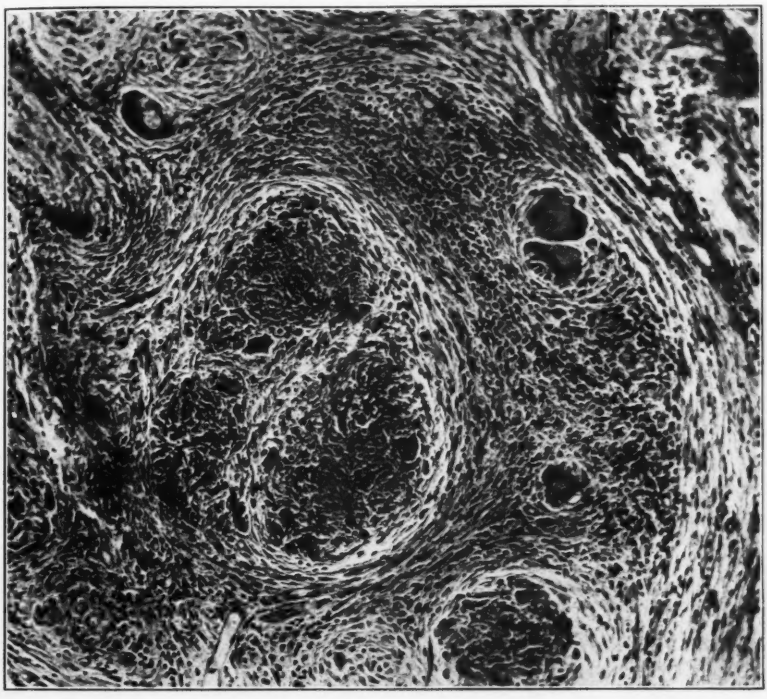

Fig. 1.—Granulomatous reaction in dermis six months after beryllium phosphor had been traumatically introduced into the tissues; hematoxylin and eosin stain; × 160.

grayish white necrotic material. The period from injury to excision varied from four months to nine years.

A fifth patient discussed here was a young woman who worked at cutting metallic beryllium with a machine and had accidentally lacerated her finger during her work. A granuloma developed, and this failed to heal over a period of four years, so that finally a complete excision of the lesion was made.

HISTOPATHOLOGIC OBSERVATIONS

The tissues which were excised four months after the phosphor-containing beryllium oxide had been introduced into them were found to be the site of a granulomatous reaction in which there were areas of

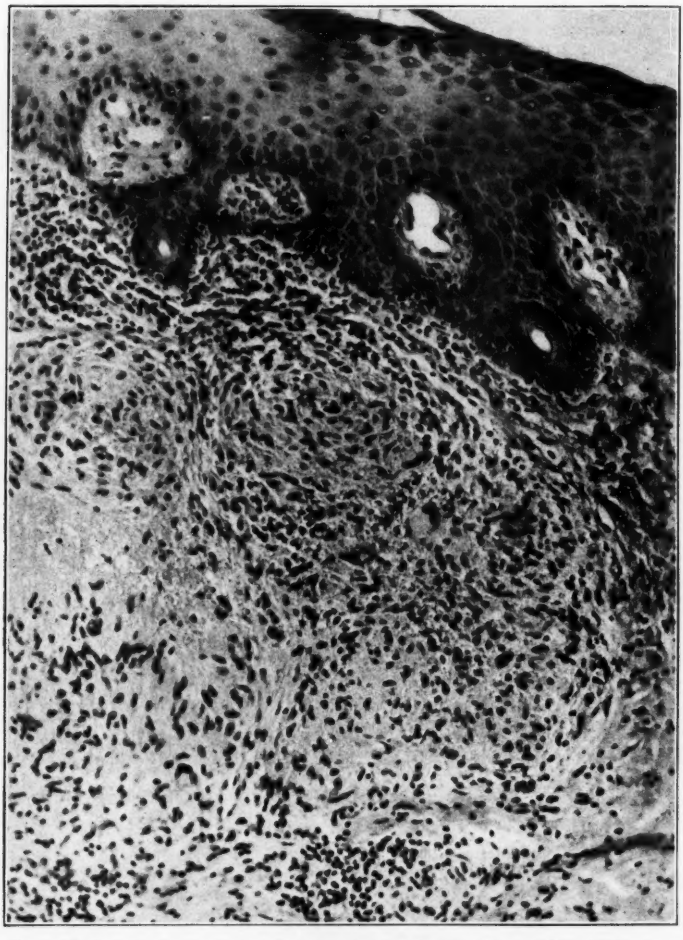

Fig. 2.—Focal necrosis and granulomatous inflammation of dermis four years after metallic beryllium had been introduced into the tissues; hematoxylin and eosin stain; × 160.

necrosis. The necrotic areas involved the dermis and subcutaneous tissues, and they were not unlike the areas of caseation in tuberculosis; they were, however, much more extensive than those in the rare cases

of Boeck's sarcoid in which caseous necrosis does occur.[4] There was amorphous, slightly eosinophilic, granular material, and at one point a small, highly refractile mass of foreign material was present. Around the necrotic center there were large mononuclear cells, some of which were polyhedral while others were spindle-shaped. These cells had large reticular nuclei, so that they resembled epithelioid cells. The necrotic areas were surrounded by a zone of partially degenerated, elongated cells resembling fibroblasts, around which there was cellular

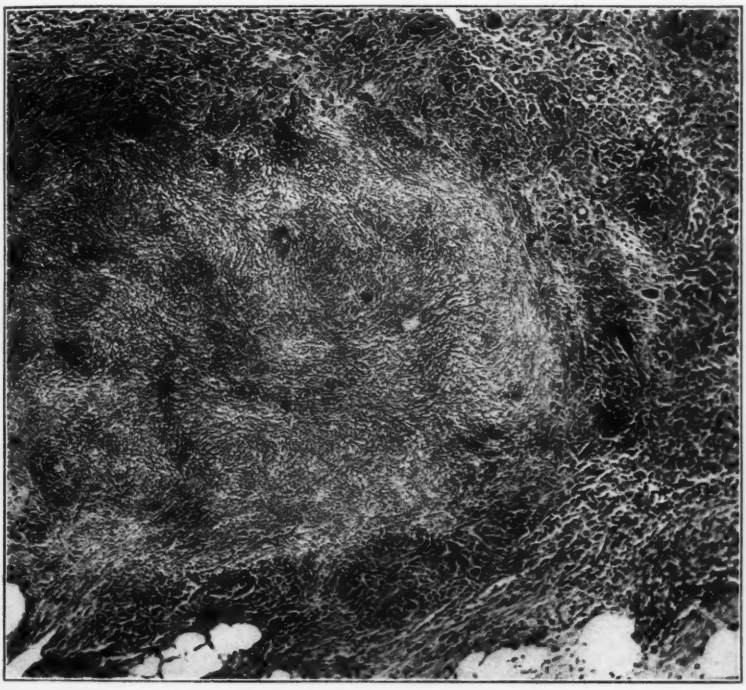

Fig. 3.—Area of necrosis of dermis four months after introduction of beryllium phosphor; hematoxylin and eosin stain; × 115.

collagenous tissue infiltrated with large numbers of lymphocytes. The remainder of the tissue was divided by relatively dense strands of connective tissue into nodules of varying sizes. The nodules were comprised in most instances of relatively loosely arranged connective tissue, in which there were elongated and polygonal cells with large reticular nuclei, surrounded by elongated fibroblasts and a few lymphocytes. The

4. Goldman, L.: Personal communication to the author.

entire nodules were surrounded by relatively dense connective tissue in circumferential arrangement. In the centers of some of the nodules and in the peripheral portions of others were multinucleated giant cells of the Langhans type, some of which were as small as 20 microns in diameter, while others were as much as 200 microns in diameter. Some of these giant cells had within their cytoplasm small masses of highly refractile substance, but in none were there inclusion bodies of the type described by Schaumann [5] or of the type described by Wolbach.[6]

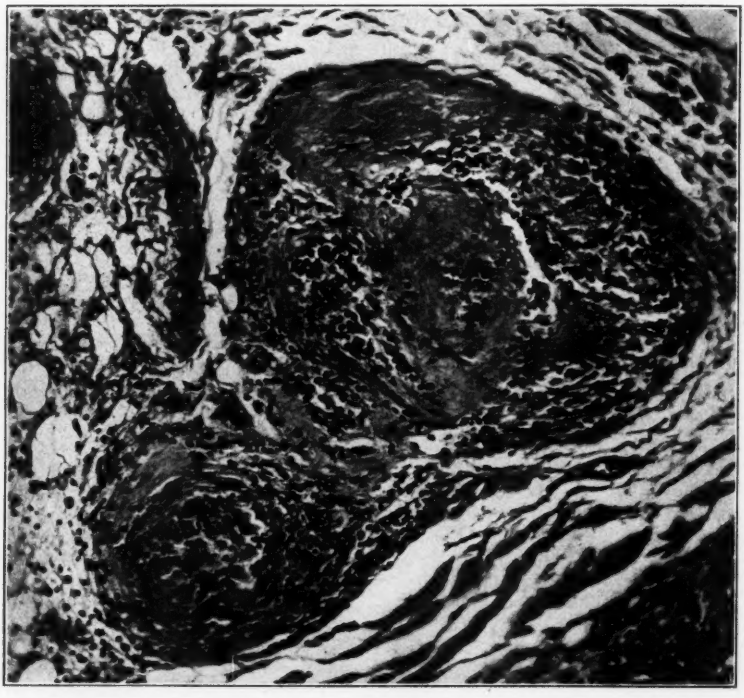

Fig. 4.—Marked fibrosis of individual nodules in granuloma nine years after beryllium phosphor was introduced into dermis; hematoxylin and eosin stain; × 160.

The lesions occupied the dermis and the uppermost portion of the subcutaneous tissues. The epidermis was intact, and there were

5. Schaumann, J.: On the Nature of Certain Peculiar Corpuscles Present in the Tissue of Lymphogranulomatosis Benigna, Acta med. Scandinav. **106**:239-253, 1941.

6. Wolbach, S. B.: A New Type of Cell Inclusion, not Parasitic, Associated with Disseminated Granulomatous Lesions, J. M. Research **24**:243-257, 1911.

moderate acanthosis, hyperkeratosis and parakeratosis. The peripheral portions of the lesions were infiltrated by numerous lymphocytes, but within the lesion itself such cells were relatively few.

The lesions from 3 other patients who had had phosphors embedded in their skin for periods of from one to four years before excision were similar to the one described. The lesion from the woman who had had metallic beryllium embedded in her skin for a period of four years was also similar to the one described.

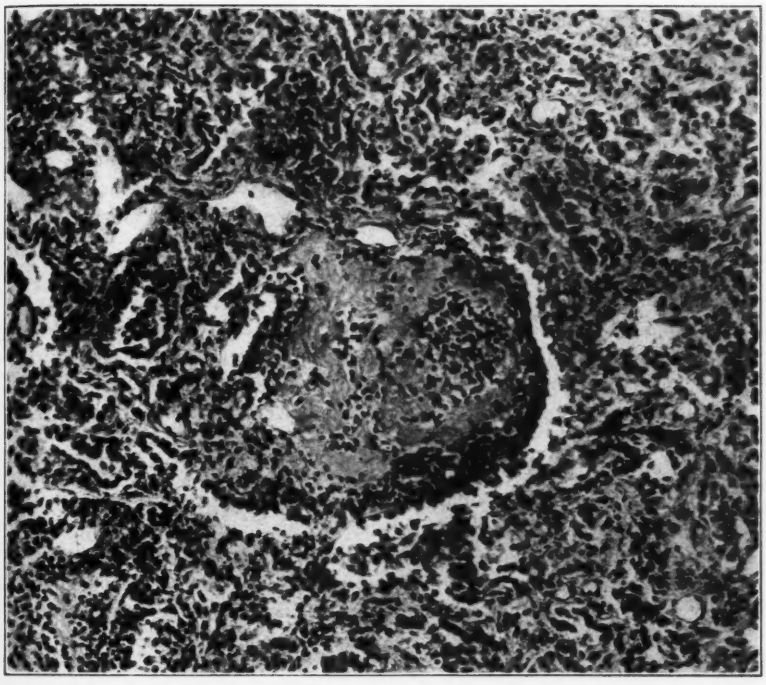

Fig. 5.—Nodule of lung comparable to those of skin shown in figure 4, in a case of chronic pulmonary berylliosis; hematoxylin and eosin stain; × 160.

The lesion in the skin of the man who had had beryllium phosphor embedded at that site nine years before it was excised differed somewhat in degree of development from those the duration of which had been four years or less. In the nine year lesion the general picture was one of dense, relatively acellular collagen in which were numerous small nodules comprised of large cells. The latter were for the most part compactly arranged, with poorly defined pale cytoplasm and large vesicular nuclei. Occasional small multinucleated cells which resembled

Langhans' cells were present. There was a large number of lymphocytes scattered throughout these nodules, interspersed between the large cells. Many of the nodules had dense collections of numerous lymphocytes in their peripheral portions, and practically all of the nodules were surrounded by collagen which contained only a few small connective tissue cells. In some places this collagen was hyalinized.

In 3 of the 5 cases sufficient amounts of tissue were available to permit chemical analysis. The tissues were analyzed for beryllium by the method of Cholak and Hubbard,[7] and small quantities of beryllium were recovered in 2 cases but could not be recovered in the third.

COMMENT

The evidence which supports the opinion that beryllium is capable of producing fibrosis has been presented in a paper [8] in which 13 cases of chronic pulmonary berylliosis with fibrosis and granulomas are described. Among 7 cases of acute pneumonitis due to the inhalation of fumes or dusts containing beryllium, there were organization of exudate and early fibrosis in 5, and the lesion progressed to the first stage of the formation of fibrous nodules in 3. Further support of the opinion that beryllium is capable of inducing fibrosis has come from experimental work in which the reactions induced by metals embedded in bone were studied.[9] It was found that a certain alloy containing nickel, cobalt, chromium and molybdenum was innocuous, but that when beryllium was added to the alloy in the concentration of 1.6 per cent, granulation tissue formed at the site of the alloy and lymphocytes infiltrated the tissues.

The diagnosis of beryllium granuloma of the skin can probably be made clinically when there is a history of beryllium or one of its compounds having been embedded in the skin. Lacerations produced by glass from fluorescent lamp tubes are likely to lead to the development of beryllium granulomas, since beryllium oxide is a constituent of the phosphorescent powders used in making these tubes. Tissues into which the material has been introduced will, over some months, show formation of excessive scar tissue, which occasionally opens and drains. Biopsy of such tissues reveals lesions which could be confused with caseous tuberculosis or Boeck's sarcoid. Differentiation rests on the presence of beryllium demonstrated by analytic methods, and on the absence of

7. Cholak, J., and Hubbard, D. M.: Spectrographic Determination of Beryllium in Biological Material and in Air, Analyt. Chem. **20**:73-76, 1948.

8. Dutra, F. R.: The Pneumonitis and Granulomatosis Peculiar to Beryllium Workers, Am. J. Path. **24**:1137-1166, 1948.

9. Campbell, E. H.; Meirowsky, A., and Tompkins, V.: Studies on the Use of Metals in Surgery: II. Experiments in the Use of Ticonium in Cranial Repair, Ann. Surg. **116**:763-775, 1942.

tubercle bacilli. The beryllium lesion can be differentiated from Boeck's sarcoid by the presence of caseous areas and by the large number of lymphocytes scattered throughout, particularly in the periphery of the lesion. It is probable that tertiary syphilis might simulate the cutaneous beryllium granuloma, and the differentiation would depend on the demonstration of treponemas and the failure to demonstrate beryllium. Nodular leprosy and certain cutaneous fungous diseases would present only minor difficulties of differentiation.

The treatment of cutaneous and subcutaneous granulomas which follow the introduction of beryllium or its compounds has been successful in the 5 cases reported here only when the tissues containing the foreign material have been completely excised. Before excision, in each case, the lesion was marked by excessive formation of scar tissue and repeated episodes of discharge of the wound, followed by temporary healing.

SUMMARY

Five cases of chronic granulomatous inflammation of the skin and subcutaneous tissues following the introduction of beryllium or beryllium compounds are presented.

The histologic relationship of the granulomas of the skin and similar granulomas which occur in the lungs of persons who have inhaled beryllium-containing dusts in industry is described.

Cure followed excision of the lesions in all cases.

"MACULES" OF LEPROSY

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HONOLULU, HAWAII

THE WORD "macule" has long been used by students of leprosy in two quite different and yet overlapping senses. Exceptionally, it has been used in the orthodox sense of a circumscribed, flat discoloration of the skin (usually hypopigmentation, and less often erythema); and in this use it is, oddly enough, usually qualified as either "lepromatous" or "simple" (or "anesthetic") or, more recently, "uncharacteristic" or "indeterminate."

The term has been much more commonly used, however, in a sense at once looser and more restricted, and certainly not at all orthodox. Without any qualifying adjective, the word "macule" [1] has been used to designate any circumscribed, discolored (hypopigmented, erythematous or both) cutaneous lesion, flat or elevated, in which (1) evidence of nerve damage (usually anesthesia) can be demonstrated and (2) bacilli are rare or absent. When such a lesion is flat, it is often spoken of as a simple or an anesthetic macule; when it is elevated (and sometimes even when it is not), it is often called a tuberculoid "macule."

There is, then, a morphologic macule, which is (by definition) flat but may occur in either the lepromatous or the tuberculoid form of leprosy, and an immunologic "macule," which may be either a (flat) macule or an elevated plaque but occurs only in the tuberculoid form of leprosy. The lepromatous macule, for reasons which will be discussed presently, occurs relatively infrequently. This fact has led to the practice of using the unqualified word "macule" almost entirely as a designation for the circumscribed, anesthetic, paucibacillary macules and plaques—the leprides of Unna—which characterize the maculoanesthetic or "neural" or, as the Fifth International Congress for Leprosy named it, the tuberculoid form of the disease.

LEPROMATOUS MACULES VERSUS "MACULES"

As Hansen observed over fifty years ago,[2] the "two forms [of leprosy] . . . are clinically pretty sharply distinguishable." This state-

From the Clinic.

Read at the Sixty-Eighth Annual Meeting of the American Dermatological Association, Inc., San Diego, Calif., April 26, 1948.

1. Quotation marks will be used throughout this article wherever the word is being used in this sense rather than in the literal dermatologic sense.

2. Hansen, G. A., and Looft, C.: Leprosy in Its Clinical and Pathological Aspects, translated by N. Walker, Bristol, J. Wright & Co., 1895.

ment is most applicable to the matured "polar" lesions; it is least true with reference to the macular lesions. It will be instructive, however, and of great practical value, for one to try to apply this observation to the simple, flat, usually hypopigmented macular lesions of the disease.

Bacterioscopy.—The bacillary content of the tissue fluid at the border of the lesion is perhaps the most useful criterion. If material obtained by Wade's "scraped incision" method [3] (which is still inaccurately but concisely called a "snip" in Hawaii) shows abundant bacilli, the lesion is probably lepromatous; if it shows few or none, the lesion is probably tuberculoid; if it shows moderate numbers, the nature of the lesion has not yet been determined, and the case, though not classifiable as either lepromatous or tuberculoid in type, may be said to be in the indeterminate group, at least so far as bacterioscopic evidence is concerned.

The examination for bacilli may also be made on sections of tissue, preferably with the aid of Fite's formaldehyde modification [4] of the Ziehl-Neelsen stain. More sensitive methods of searching for bacilli (as described by de Souza-Araujo [5]) are useful in diagnosis but not particularly helpful in differentiating the types.

Neurologic Findings.—Concomitant and coextensive nerve damage is probably of almost equal importance in classifying the case as tuberculoid. Demonstrable anesthesia which is identical in extent with the hypopigmentation is strong evidence that the lesion is not lepromatous but maculoanesthetic, i. e., tuberculoid in immunologic type if not (yet) in histologic structure, for it is in this type that the neurolytic defensive reaction to the bacillus occurs. In this connection, it is often forgotten even by experienced workers that anesthesia in leprosy is apt to occur in a dissociated pattern; e. g., anesthesia to cold or heat or to both, with unimpaired sensitivity to light touch and to pinprick, is commonly found in early lesions. In lesions manifesting no apparent anesthesia, the histamine test of Rodriguez and Plantilla [6] may be employed (in patients with light-colored skins) to detect lesser degrees of nerve dam-

3. Wade, H. W.: Bacteriological Examination in Leprosy, Leprosy Rev. **6**:54 (April) 1935.

4. Fite, G. L.: The Staining of Acid-Fast Bacilli in Paraffin Sections, Am. J. Path. **14**:491 (July) 1938; The Fuchsin-Formaldehyde Method of Staining Acid-Fast Bacilli in Paraffin Sections, J. Lab. & Clin. Med. **25**:743 (April) 1940. Tilden, I. L., and Tanaka, M.: Fite's Fuchsin-Formaldehyde Method for Acid-Fast Bacilli Applied to Frozen Sections, Am. J. Clin. Path. **15**:95 (Nov.) 1945.

5. de Souza-Araujo, H. C.: Clamp Method to Obtain Cutaneous Lymph in the Diagnosis of Leprosy, Leprosy Rev. **18**:44 (April-July) 1947.

6. Rodriguez, J., and Plantilla, F. C.: The Histamine Test as an Aid in the Diagnosis of Early Leprosy, Philippine J. Sc. **46**:123 (Sept.) 1931; Internat. J. Leprosy **1**:49 (Jan.) 1933.

age; in darker skins the sweating tests described by Muir [7] and Degotte [8] (using intradermally administered pilocarpine or exercise) or by Myerson [9] and his associates (using methacholine), which seem to be just as sensitive, may be used to better advantage.[10] The important thing is the demonstration of nerve damage which is coextensive with the visible macule; this is highly characteristic of the maculoanesthetic—the "neural" or tuberculoid—form of leprosy.

Clinical Aspects.—The remaining clinical features by which one may judge the hypothetic macule constitute much less useful criteria of its type. The classic lepromatous macule tends to be multiple, and the tuberculoid, solitary, but many exceptions occur. The lepromatous macule generally has a poorly defined border, often with fine pseudopod-like irregularities; the tuberculoid macule is usually sharply outlined. By the same token, lepromatous macules tend to confluence and tuberculoid ones to discreteness. Lepromatous macules are likely to occur on the trunk; a macule on the face or extremities is more commonly of the tuberculoid variety. The lepromatous macule usually persists for many months or alternatively progresses into the clinical lesion known as a leproma; the anesthetic macule is likely to show evidence of regression within six months to a year. None of these findings, however, is conclusive, and if the bacterioscopic and neurologic criteria are inconsistent one must resort to biopsy. Indeed, biopsy should be done anyway, as a routine precaution.

Histologic Changes.—Histologic changes in such early and minimally advanced lesions as the macules being discussed are pretty likely to be banal, so far as the general pathologist's interpretation of them is concerned. Ermakova, of Moscow,[11] found them so in a considerable series of anesthetic flat "macules." Wade,[12] on the other hand, found changes which he regarded as "nearly always in some degree tubercu-

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7. Rogers, L., and Muir, E.: Leprosy, ed. 3, Baltimore, Williams & Wilkins Company, 1946.

8. Degotte, J.: Practical Application of Sweating Dysfunction to the Diagnosis of Leprosy, Rec. tr. sc. méd. Congo, January 1942, p. 135.

9. Myerson, A.; Loman, J., and Rinkel, M.: Human Autonomic Pharmacology: VI. General and Local Sweating Produced by Acetyl-Beta-Methyl Choline Chloride (Mecholyl), Am. J. M. Sc. **194**:75 (July) 1937. Arnold, H. L., Jr.: The Sweat Response to Intradermally Injected Mecholyl: Preliminary Report of Its Possible Use in the Diagnosis of Leprosy, Proc. Staff Meet. Clin., Honolulu **11**:75 (Aug.) 1945.

10. Arnold, H. L., Jr.: The Intradermal Mecholyl Test for Anidrosis; A. Diagnostic Aid in Leprosy, Internat. J. Leprosy **16**:335 (July-Sept.) 1948.

11. Ermakova, N.: The Histopathology of Simple Leprids, Internat. J. Leprosy **7**:495 (Oct.-Dec.) 1939.

12. Wade, H. W., and Rodriguez, J. N.: The Skin Lesions of Early Leprosy: II. Observations in Cebu, Internat. J. Leprosy **5**:1 (Jan.-March) 1937.

loid" in a larger series of similar lesions. He often found it necessary to employ serial sections, however. Ota and Sato,[13] in Japan, came independently to exactly the same conclusion in the same year.

What is meant by "in some degree tuberculoid"? Not fully developed, noncaseating, epithelioid cell tubercles, to be sure. In general, the designation refers to an infiltrate of rather sharply focal arrangement, composed of both lymphocytes and epithelioid cells, which may or may not be arranged in the pattern of definite tubercles. Diffuseness or vague margination of the infiltrate and scarcity of lymphocytes are both suggestive of the lepromatous form of the disease.

THE INDETERMINATE GROUP

If bacterioscopic, neurologic, clinical and histologic changes are inconclusive or inconsistent, the problem is a more difficult one; it may indeed be incapable of solution for the time being. The South American dermatologists, perhaps impatient at the postponement of an exact diagnosis, have urged the establishment of a third "type" or "form" of leprosy to include all such cases: they suggested calling them *incaracterístico* (uncharacteristic). At the Fifth International Congress for Leprosy, in April 1948, the words "indeterminate" and "group" were agreed on: the former because it permitted the use of the same initial letter, *I*, in Portuguese, Spanish, French and English, and the latter to indicate that this term designated, not a third type of leprosy, but only a group of cases, defined largely by their lack of definitive characteristics. It has been shown that such cases may remain in this neutral status for a variable period, at the end of which they will heal, become lepromatous or become tuberculoid.

In addition to the differentiating criteria which have been discussed, there is one more point, regarded by some workers as the most informative and important of all, which presents so many ramifications that I shall discuss it under a separate heading.

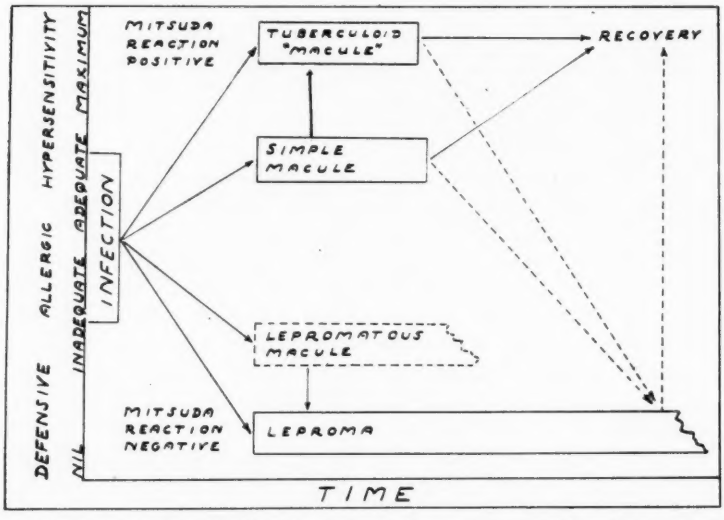
LEPROMIN (FERNÁNDEZ AND MITSUDA) REACTIONS

Kensuke Mitsuda reported in 1916[14] that the intracutaneous injection of a small amount of a phenolized, boiled suspension of finely divided lepromatous granulation tissue (a mixture of dead Mycobacterium leprae and human tissue) would produce a slowly developing often ulcerated papule in the great majority of patients with the "maculoanesthetic" (i. e., tuberculoid) form of leprosy, as well as in many

13. Ota, M., and Sato, S.: Tuberculoid Changes in Leprosy, Internat. J. Leprosy **5**:199 (April-June) 1937.

14. (*a*) Mitsuda, K., in discussion in Jap. J. Dermat. **16**:513 (June) 1916; (*b*) cited by Hayashi, F.: Mitsuda's Skin Reaction in Leprosy, Internat. J. Leprosy **1**:31 (Jan.) 1933.

presumably uninfected persons; it regularly produced little or no reaction in the great majority of patients with the "tubercular" (the nodular, or lepromatous) form. He extended and amplified his observations in a series of reports, which received little attention in this country until the publication of a paper by his student, Hayashi, in 1933.[15] His observations have since been widely confirmed. Numerous studies have been made in an effort to improve and standardize the test by purification and fractionation of the test material. These have not been especially rewarding, however, since the crude preparation seems as potent as, and no less specific than, its various fractions.[16]



Diagrammatic oversimplification of the major cutaneous lesions of leprosy as functions of (1) "defensive allergic hypersensitivity" to the bacillus and (2) time. Note that this hypersensitivity may already exist at the time of infection, that it may either increase or decrease after infection occurs, that it may be demonstrated by a positive Mitsuda reaction, that it results, in general, in self limited lesions known as "macules," that its failure to develop results in essentially persistent or progressive lesions and that it rarely, if ever, develops late in the course of the disease. Note, too, that exceptionally this hypersensitivity may be lost, resulting in transition of the disease to the lepromatous form and that, also exceptionally, recovery will occur in a lepromatous case, without, however (or sometimes with?), transition through a tuberculoid phase.

The term "defensive allergic hypersensitivity" is to be taken loosely, not literally. And the prognosis as indicated does not take into account the effect of treatment with sulfone derivatives.

15. Hayashi.[14b]

16. Davey, T. F.: Some Observations on the Role of Allergy in Leprosy, Leprosy Rev. **17**:42 (July); 75 (Oct.) 1946.

Fernández,[17] in 1940, reported that the often observed tuberculin type of reaction to lepromin, reaching peak severity in forty-eight hours instead of the two to five weeks required for the Mitsuda reaction, could be correlated with the clinical form of the leprosy virtually as closely as could the latter. Dharmendra and Lowe,[18] in 1943, showed that this response could be elicited regularly by injection of the purified protein, presumably the nucleoprotein, of the bacilli. The injection of the crude antigen, on the other hand, generally produced relatively little effect until the bacilli had had time to break down and liberate this antigenic material. It is highly probable, at all events, on the basis of this evidence and the analogy with some (though by no means all) forms of tuberculosis, that a positive lepromin test indicates acquired allergic hypersensitivity to the antigens of the leprosy bacillus, though this may have been acquired without infection by, or even contact with, the organism in question. I know of no student of leprosy who believes that a strongly positive Mitsuda reaction can occur in a patient with low resistance to leprosy, or a negative or even a feeble reaction in one with high resistance. The allergic response and the defensive one seem, in leprosy, inseparable.

Hayashi [14b] and others showed that the Mitsuda reaction may be more pronounced in tuberculoid "macules" than in normal skin. This observation was confirmed most recently by Davey.[16] The reaction is apt to be stronger in elevated tuberculoid "macules" than in flat ones and is usually strongest in their borders. A positive reaction is also occasionally found, according to Davey, in lepromatous macules, even when the normal skin outside the macule gives a negative reaction. Dharmendra,[19] in a recent extensive review of the literature on the lepromin test, stated that he had not been able to confirm this observation, and my experience in Hawaii has been similar.

SIMPLE (FLAT) MACULES VERSUS TUBERCULOID (ELEVATED) "MACULES"

I have discussed in some detail the features of the two types of flat macules, the lepromatous and the anesthetic, or simple. The elevated, or tuberculoid, "macules" present no such highly refined problem in differentiation, for in them the allergic hypersensitivity to Myco. leprae is strongly and usually securely established. The histologic changes, which are almost invariably of the tuberculoid pattern, with

17. Fernández, J. M. M.: The Early Reaction Induced by Lepromin, Internat. J. Leprosy **8**:1 (Jan.-March) 1940.

18. Dharmendra and Lowe, J.: Studies of the Lepromin Test, Leprosy in India **15**:82 (April) 1943; Leprosy Rev. **17**:9 (April) 1946.

19. Dharmendra: The Lepromin Test: A Review, Leprosy Rev. **18**:92 (Oct.) 1947.

well defined, noncaseating, epithelioid cell tubercles and no or comparatively few bacilli, bear out this statement, in accordance with the observations of Jadassohn and Lewandowsky. It seems probable that in these lesions there is elaboration of abundant antibody by the histiocytes in the skin, and a resultant antigen-antibody reaction which effectively eliminates the bacilli. Why this inflammatory reaction is rarely accompanied with necrosis when it occurs in the skin, and is usually accompanied with it when it occurs in the nerves, is not clear; but this finding has often been reported,[20] and my observations in Hawaii have regularly confirmed it. Of course this necrosis of nerve tissue is the reason for the regular occurrence of anesthesia or anhidrosis in even the earliest "macules," and it has led to the confusion begun by Danielssen and Boeck[21] (and checked only for a time by Hansen and Looft[2]), resulting from the designation "anesthetic" or "nerve" or "neural" for cases of this form of the disease and also for lepromatous cases with more nerve damage than cutaneous damage.

It may be significant that there is not necessarily any difference between this tuberculoid response, produced by presumably living Myco. leprae, and a positive Mitsuda reaction, produced by dead ones; for, indeed, as Davey[16] pointed out, if the former happens to be vigorous and intense enough, or to be accompanied by sufficiently pronounced fixation of the bacilli in the tissues, it may run the same swift and benign course as the Mitsuda test, with final complete resolution in a few weeks or months. Ordinarily, however, the multiplication of bacilli, ahead of (or in) the advancing border of the lesion, is able to keep slightly ahead of the development of the defensive tissue reaction, so that gradual spread occurs, and only after some months, or even longer, does either the "anchoring" of the bacilli or the more effective production of antibodies permit all the organisms to be destroyed, so that the lesion heals. Healing often occurs in the central zone and spreads peripherally, so that annular lesions from a few millimeters to several decimeters in diameter are formed, and "cockade" patterns, produced by successive fluctuations in the vigor of the antigen-antibody reaction, are not unusual. Such patterns are not infrequently observed in patients whose disease is undergoing transition from the tuberculoid to the lepromatous type.

The effectiveness and persistence of this combined defensiveness and hypersensitivity may vary widely. Transitory relapses after complete or partial healing of the lesions are not rare. Even gradual, persistent

20. Ota and Sato.[18] Pardo-Castello, V.; Tiant, F. R., and Piñeyro, R.: Nerve Lesions of Leprosy, Arch. Dermat. & Syph. **55**:783 (June) 1947.

21. Danielssen, D. C., and Boeck, C. W.: Traité de la spédalskhed ou éléphantiasis des grecs, Paris, J.-B. Baillière, 1848.

loss of hypersensitivity, with resultant reversal of the lepromin reaction to negative and transformation of the lesions from tuberculoid to lepromatous, may occur, though it is so infrequent that some workers have never observed it and some, indeed, doubt that it occurs. I have seen it occur twice, both times in patients with large and numerous "major tuberculoid" lesions. The first alarming sign was the tendency of both patients to have frequent exacerbations of the cutaneous lesions (tuberculoid reactions). Wade [22] described this process in detail in a few cases. The reverse of this phenomenon of transition (i. e., a change from the lepromatous to the tuberculoid type) seems to be much rarer, presumably because the patient will usually develop hypersensitivity and resistance to the bacillus early in the course of his infection if he is capable of developing them at all. The only definite evidence that transformation in this direction can occur, in fact, was presented recently by Lauro de Souza Lima.[23]

TUBERCULOID "MACULES" VERSUS LEPROMAS

In considering tuberculoid "macules" in contrast with lepromas, one is dealing with the easily distinguished "polar" lesions of the disease: the two extremes, one representing victory for the patient's defensive processes and the other representing victory for Myco. leprae. All that was said about the differentiation of the lepromatous macule from the simple or anesthetic macule may be applied here. The tuberculoid "macule" (whether macule or plaque) is regularly anesthetic; the leproma is anesthetic only if (as occasionally happens) it occurs in an anesthetic area of skin. The tuberculoid "macule" typically contains few or no bacilli; even in reactions, it rarely contains the abundance of bacilli found regularly in a leproma. The tuberculoid "macule" characteristically shows noncaseating, epithelioid cell tubercles, with abundant lymphocytes, histologically; the leproma has the architecture and composition of a fatty foreign body granuloma, or xanthoma, in which the lipid material is presumably furnished by the masses of Myco. leprae, and in which lymphocytes are few.

Clinical features, as with the flat macules, are less trustworthy; however, like flat tuberculoid macules, tuberculoid plaques are somewhat more likely to be solitary, or few, and asymmetric, and lepromas, generalized and symmetrically distributed. Lepromas tend to develop earliest and most conspicuously on acral parts: ears, forehead, chin, buttocks, elbows and fingers. Tuberculoid lesions show little or no

22. Wade, H. W., and Rodriguez, J. N.: Borderline Tuberculoid Leprosy, Internat. J. Leprosy **8**:307 (July-Sept.) 1940.

23. Lima, L. de S., and de Souza Campos, N.: Immuno-Biologic Anomalies in Leprosy, Internat. J. Leprosy **16**:9 (Jan.-March) 1948.

real predilection for these parts. A sharply defined and elevated border or a clearing center is much more likely to be manifested in a tuberculoid lesion than in a leproma, though the sharply clear center, with a less clearly defined outer margin, suggests the leproma rather than the tuberculoid lesion. Norman Sloan [24] pointed out that lepromas have a strong tendency to respect facial and other natural grooves and creases, such as the nasolabial fold and the angle between the root of the nose and the inner corners of the eyes; a tuberculoid plaque will often invade and involve such areas. Involvement of the mucous membrane (of the eye, mouth, nose, throat and larynx) and testicular involvement occur only in lepromatous cases. Nerve involvement occurs regularly in both lepromatous and tuberculoid cases, but nerve damage is produced only slowly by the gentle, passive, bacteriophagic lepromatous response, whereas it is produced early and rapidly by the vigorous, defensive, bacteriolytic tuberculoid response.

SUMMARY AND CONCLUSIONS

The flat macules and elevated "macules" of leprosy apparently represent circumscribed cutaneous areas of acquired allergic hypersensitivity to the nucleoprotein of Myco. leprae.

If this hypersensitivity is lacking or inadequate to destroy the organisms, the macule is called a lepromatous macule, and it can usually be identified as such by the abundance of bacilli in it and its failure to manifest anesthesia or other evidence of nerve damage.

If this hypersensitivity is adequate to destroy the organisms, the lesion is then often spoken of simply as a "macule" (or, better, a tuberculoid plaque), and it can usually be recognized by its relative lack of bacilli and the regular occurrence of evidence of nerve damage throughout its visible extent. If such lesions are flat they are sometimes called "simple macules" or "anesthetic macules"; if they are elevated they are usually called "tuberculoid macules." Both terms were included under Unna's word "leprides."

The development of an adequate degree of resistance and allergic hypersensitivity to Myco. leprae results in the establishment of a relatively favorable form of leprosy which has been known as the "anesthetic" (Danielssen, 1847), "maculoanesthetic" (Hansen, 1895), "neural" and, more recently, "tuberculoid" form.

The failure of development of such a state following infection with Myco. leprae results in the establishment of an unfavorable form of leprosy known as the "tubercular" (Danielssen), "nodular" (Hansen), "cutaneous" and, more recently, "lepromatous" form.

24. Sloan, N. R.: Personal communication to the author.

Such hypersensitivity, once developed, usually results in eventual spontaneous cure of the infection; it may, however, be lost, with resultant transition of the case from the tuberculoid to the lepromatous category.

The lepromin test appears to be a fairly trustworthy method of measuring this hypersensitivity, both in normal skin and in macules.

The Clinic, South Hotel Street at Thomas Square (13).

ABSTRACT OF DISCUSSION

DR. BRAULIO SÁENZ, Habana, Cuba: The last time our Association met on the Pacific coast was in 1938, at Del Monte, Calif. At that meeting I presented a paper on tuberculoid leprosy (ARCH. DERMAT. & SYPH. **39**:456 [March] 1939), the first on this subject in North America. The paper was written prior to the holding in Cairo of the Fourth International Congress for Leprosy and was read shortly thereafter. It was at that congress that the South American classification of leprosy was rejected and the Cairo classification adopted.

My paper, being a pioneer one and based on a limited number of observations, was no doubt incomplete and not devoid of some minor erroneous interpretations. Ten years later, on April 10, 1948, the Fifth International Congress for Leprosy took place in Habana, and the South American classification (now known as the classification of Habana) was adopted. Dr. Arnold has very aptly outlined his paper on this classification, which, for the time being, is the only one tenable, because it is based on the following points: (1) the morphology, or clinical aspect of the lesions, (2) immunologic reactions, (3) bacteriologic criteria and (4) a more or less definite histopathologic picture.

The presenter stresses the importance of these fundamental bases and discusses the position of the indeterminate groups, previously called uncharacteristic. This, as he indicates, may be the possible weak point of this classification, but one must remember that, not only in leprosy, but also in many other dermatologic processes, only the evolution determines the real position of certain cases and that borderline cases are often observed. In addition, this group, in the majority of cases, presents a negative reaction to the Mitsuda test, which is a possible indication of future transition to the lepromatous form.

The publications of F. Fischl (*Dermat. Ztschr.* **55**:274 [March] 1929), and of Jadassohn (ARCH. DERMAT. & SYPH. **21**:355 [March] 1930) and the communications of J. Darier, Civatte, P. Woringer and H. Gougerot to the International Dermatological Congress at Budapest in 1935 (Deliberationes Dermatologorum Internationalis, IX-1, Budapest, September 1935, Leipzig, Johann Ambrosius Barth, 1936), made important contributions explaining the role of the biologic reactions of immunity on the morphologic aspects of the cutaneous lesions; nevertheless, some points remain unanswered. For example, some have denied the possibility of the transformation of the tuberculoid lesions to the lepromatous form; despite such denial, this transformation is an established fact. Dr. Arnold has observed 3 such cases, and I have seen 4. Some three months ago, I requested Dr. Nelson Souza Campos, who has had considerable experience in the treatment of patients in the Sanatorio Padre Bento, São Paulo, Brazil, for his opinion on the subject. He replied to the effect that he had observed 64 cases of tuberculoid leprosy of the reactional type which had undergone transformation to the lepromatous type. All cases were observed in patients in leprosy asylums. The possible explanation is that these patients had been superinfected from other lepromatous patients. This explanation did not apply to my patients, who were all encountered in my private

practice. Therefore, I should like to ask Dr. Arnold whether his cases were encountered in an asylum or in private practice. Through the action of the sulfone drugs, the reverse of this transformation was reported to the Fifth International Congress for Leprosy by Lauro de Souza Lima (*Internat. J. Leprosy* **16**:127 [April-June] 1948): Lepromatous leprosy was transformed into the tuberculoid type. The histopathologic changes from one form to the other were checked at different dates, the tissular changes appearing first as the expression of an acquired immunity; later the reaction to the Mitsuda test became positive, and as a final stage came the curability of some of the cases. Before closing I wish to congratulate Dr. Arnold heartily on his timely paper; it is simple and concise, and it gives complete descriptions of the polar and of the indeterminate forms of leprosy, as well as valuable information to the dermatologist respecting correct diagnosis, prognosis and epidemiology of the disease.

I also wish to express my satisfaction for the opportunity I have been given to open the discussion on this excellent contribution; in addition, it gives me pleasure to inform Dr. Arnold that I have been requested to advise him that he has been proposed as a correspondent member of Sociedad Cubana de Dermatologia y Sifilografia, and of Academia Espanola de Dermatologia y Sifilografia, as a reward for his qualifications as a dermatoleprologist.

Dr. Louis Winer, Los Angeles: I wish to compliment Dr. Arnold on his excellent paper. My associates and I have had such cases on two occasions in Los Angeles. These cases histologically were definitely tuberculoid in structure. On use of the Ziehl-Neelsen stain, numerous acid-fast bacilli were observed. The reaction to the lepromin test was negative. Because of these observations we were unable to classify the patients. However, the indeterminate group provides a classification modification, so that now these patients can be classified. I think that the histologic characteristics of tuberculoid formation are indicative that a better prognosis can be expected than if these characteristics were not present.

Dr. Hamilton Montgomery, Rochester, Minn.: I should like to ask Dr. Arnold and Dr. Sáenz about recent reports in the literature regarding the reaction to lepromin in patients with sarcoid and tuberculosis. It would appear that in sarcoidosis there is a negative reaction, whereas in active pulmonary tuberculosis the reaction may be positive. If such observations are substantiated, the lepromin test might be of diagnostic value in northern climates where leprosy is not endemic in distinguishing between an annular sarcoid and a tuberculoid leprosy. Both show an epithelioid tuberculoid reaction histologically, so that in older literature in Scandinavian countries the question was raised as to whether the patient had sarcoid or tuberculoid leprosy. Tuberculoid leprosy, I understand, gives a positive lepromin or Mitsuda reaction.

Dr. Fred D. Weidman, Philadelphia: I should like to ask Dr. Arnold whether he is able to get a supply of *Nachtblau,* which Dr. Pardo-Castello told us about and which is superior to the ordinary Ziehl-Neelsen technic for staining the bacilli.

I have tried to secure a supply, without success. Does he think that the Fite technic is also superior?

Dr. Harry L. Arnold Jr., Honolulu, Hawaii: I am obliged to all the discussers. Dr. Sáenz asked whether the transitional cases were private or asylum cases: they were all asylum cases, 1 in Mexico and 2 in Hawaii, and all showed typically tuberculoid annules with the "cockade" pattern, with numerous bacilli and (in those seen in Hawaii, at least) a negative reaction to the lepromin test. Dr. Winer's cases with tuberculoid lesions with numerous bacilli may be examples of

this "transitional" variety; they may merely be cases undergoing a single transitory tuberculoid reaction. In reply to Dr. Montgomery's query regarding sarcoid: I was not aware that the lepromin test was necessarily negative in sarcoid, or at least in sarcoid not due to leprosy. It had been my feeling that in areas where leprosy was not endemic, positive reactions to lepromin would be relatively unusual and that many cases of sarcoid would therefore, in such areas, be fairly likely to have negative Mitsuda reactions—permitting tuberculoid leprosy to be excluded with fair accuracy. This situation does not exist in Hawaii, or in any place where leprosy is endemic. However, if anergy in sarcoid is expressed in a negative reaction to lepromin as well as in a negative reaction to tuberculin, the problem is greatly simplified. In answer to Dr. Weidman, I believe that *Nachtblau* cannot be purchased at the present time, but Dr. Reenstierna has promised to give me a small quantity of it, and perhaps some more can be obtained through him at the University of Uppsala in Sweden. As to the Fite technic, Fite himself has virtually abandoned it in favor of the Feraca procedure, which is certainly far simpler and more rapid and which he believes is equally satisfactory for finding rare organisms. My colleague Dr. Tilden and I still prefer to see blue-black bacilli against a pale orange-yellow background.

FATE OF CENTRAL NEVUS IN LEUKODERMA ACQUISITUM CENTRIFUGUM

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MOST accounts of leukoderma acquisitum centrifugum do not make clear what, if anything, ultimately happens to the central hyperpigmented spots that make this condition so distinctive. Neither Sutton,[1] who originally described the picture minutely after Hyde [2] had noted it, nor Leider and Cohen [3] in their recent review discussed the eventual disposition of the moles. However, a few of the intervening observers,[4] particularly in the foreign literature, remarked that the central pigmentations sometimes disappear. To quote one, Kuske[4c] wrote that " . . . one gets . . . the impression, from history and objective finding, that in some of the lesions undergoing enlargement, the nevus in the center is gradually obliterated by the depigmenting process . . ." [5]

In 1 of Sutton's 2 original cases, the mother of the patient (a child), stated that the central spot had not been present before the onset of the depigmentation. In consequence of such observations on the part of patients or their close relatives, some authors have theorized that the pigmented areas in the centers form because melanin, or propigment, drains from the periphery into a centripetal concentration. We doubt that these observations are correct and that the resultant theory is

1. Sutton, R. L.: An Unusual Variety of Vitiligo (Leukoderma Acquisitum Centrifugum): Report of Two Cases, J. Cutan. Dis. **34**:797, 1916.

2. Hyde, J. N.: Vitiligo with a Central Mole, J. Cutan. Dis. **24**:54, 1906.

3. Leider, M., and Cohen, T. M.: Leukoderma Acquisitum Centrifugum, Arch. Dermat. & Syph. **57**:380 (March) 1948.

4. (*a*) Gougerot, H., and Carteaud, A.: Maladie de Sutton avec régression d'un naevus pigmentaire pendent l'accroissement de l'achromie, Bull. Soc. franç. de dermat. et syph. **41**:1526, 1934. (*b*) Meirowsky, E.: Leukoderma Acquisitum Centrifugum (Sutton), in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten Berlin, Julius Springer, 1933, vol. 4, pt. 2, p. 1000. (*c*) Kuske, H.: Ueber Leukoderma acquisitum centrifugum (Sutton), Dermatologica **88**:282, 1943.

5. The original read ". . . bekommt man . . . durch die Anamnese und durch den objectiven Befund den Eindruck dass bei einigen sich ausdehnenden Elementen auch der Naevus im Zentrum allmählich dem Depigmentierungsprozess verfällt . . ."

true. Nevertheless, the possibility led Niles [6] to attempt cure of the condition by extirpation of the central spots in the hope of interrupting the assumed attraction of pigment thereto. Niles reported failure to influence the depigmenting process by this means. Nor did the central pigmentations tend to recur, as one would expect they possibly would if the theory of centripetal drainage of pigment had any virture. Moreover, in undisturbed cases, the central nevi or hyperpigmentations, according to this theory, should grow larger for as long at least as the process is active. This situation we believe and will show does not exist.

Leider and Cohen,[8] among many others before them,[7] maintained that leukoderma acquisitum centrifugum is nothing more than banal vitiligo that starts accidentally, or for the particular but unknown reason that causes all depigmentation, around preexisting moles. Kuske,[4c] who was previously quoted, expressed disagreement in the following words: ". . . Sharply delimited, round or oval, depigmented spots without a visible nevus can readily be confused with true vitiligo . . . At present it appears to me that a distinction [between Sutton's phenomenon and vitiligo] is justifiedly made . . ."[8] Kuske based his conviction on a table of clinical differences which we do not find persuasive.

In support of the contention that leukoderma acquisitum centrifugum is merely a variant of simple vitiligo, we report by photographs the following instances of spontaneous disappearance of the central maculopapules while the patients were under prolonged observation. We never saw nevi come into being, nor could the patients assert that the nevi had not been present long before the depigmentation had begun. We noted established vitiliginous areas increase in size both centrifugally and centripetally, i. e., away from and into the hyperpigmented centers, and we saw new depigmentations begin around other old nevi. The nevi seemed to be invaded by the depigmenting process and were gradually blotted out. We can make nothing more of this phenomenon than the usual progression of vitiligo. The central hyperpigmented areas persist only so long as it takes the depigmenting mechanism, whatever it may be, to bleach them completely. The reason that it takes as long as it

6. Niles, H. D.: Leukoderma Acquisitum Centrifugum, Arch. Dermat. & Syph. **43**:357 (Feb.) 1941.

7. (*a*) Sutton.[1] (*b*) Hyde.[2] (*c*) Hebra, F., and Kaposi, M.: On Diseases of the Skin, Including the Exanthemata, translated and edited by W. Tay, London, The New Sydenham Society, 1874, vol. 3, p. 180. (*d*) Stokes, J. H.: Leukoderma Acquisitum Centrifugum (Sutton), Arch. Dermat. & Syph. **7**:611 (May) 1923.

8. The original read ". . . Scharf begrenste, runde oder ovale, depigmentierte Herde ohne sichtbaren Naevus könnten leicht mit echter Vitiligo verwechselt werden . . . Zur Zeit scheint mir die Trennung gerechtfertigt . . ."

does for the central pigmentation to disappear is perhaps that the amount of pigment is so excessive or that nevi resist withering on account of their inherent good capacity to produce pigment. Also, it seems to us that the very disappearance of the central pigment in this manner disposes of theories like those of Feldman and Lashinsky [9] and others that leukoderma acquisitum centrifugum is a nevoid disease *sui generis*. If one wishes to admit vitiligo as a nevoid anomaly (tardus), then leukoderma acquisitum centrifugum is a nevus, too, but still a variant of vitiligo.

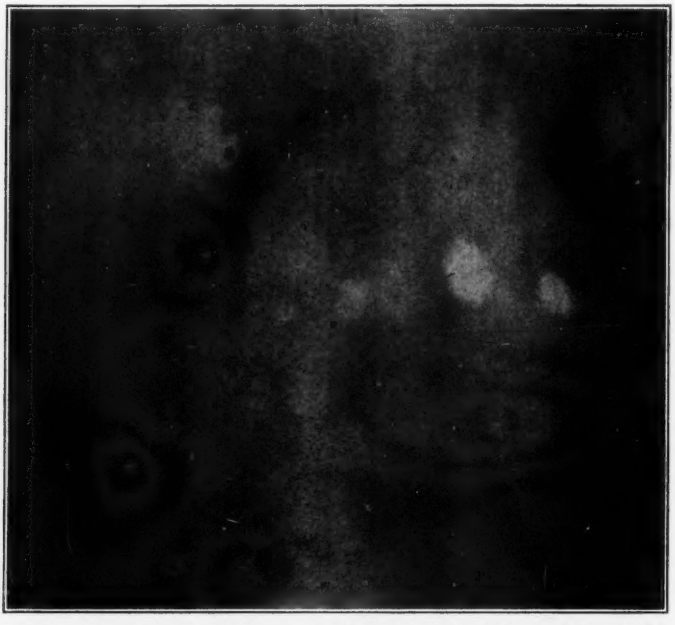

Fig. 1.—Leukoderma acquisitum centrifugum, showing lesions with and without central pigmented spots. What appears to be residual pigment in some instances is erythema.

Figure 1 shows comparable circles of vitiligo, some bearing the central pigmented spots and some lacking them. When the patient was first seen by one of us (A. A. F.), almost all lesions bore a nevus. Many of these nevi are known, from periodic examination at regular intervals over several years, to have vanished, leaving only the enlarging vitiliginous areas. In this case, histologic examination of a site which was

9. Feldman, S., and Lashinsky, I. M.: Halo Nevus: Leukoderma Centrifugum Acquisitum (Sutton); Leucopigmentary Nevus, Arch. Dermat. & Syph. **34**:590 (Oct.) 1936.

marked to have had a preexisting nevus at one time, but from which a biopsy specimen was taken after spontaneous depigmentation had occurred, showed absence of nevus cells and only the negative picture of vitiligo. When the central spot is an indisputable nevus rather than an ephelis or lentigo, another nice question is that of what becomes of the nevus cells on disappearance of the mole. Are they destroyed, or are they dispersed? However that may be, we think that any person with as many lesions as these, some adorned with and some bare of

Fig. 2.—Leukoderma acquisitum centrifugum. Compare with figure 1.

the central spot, may be assumed to have had more central pigmented maculopapules to begin with, some of which had disappeared by the time the first examination was made. This must have been the situation for the patient in case 1 of Leider and Cohen (fig. 2), whose photograph shows a nearly identical picture.

Figures 3 and 4 are "before" and "after" photographs of the patient in case 2 in the article of Leider and Cohen. The spontaneous dissolution of the central preexisting nevus was complete. What appears in the picture as a pigmented center was erythema, which photographed

dark. In the flesh it was red and it would show so on a color photograph. The interim was eighteen months, and during this period the gradual progressive depigmentation had been noted on several occasions. In both of these cases in which we were able to follow the extinction of the nevi, we saw that as the pigmentation faded it left erythema, which then eventually blanched and blended into the surrounding pallor of the vitiligo. In addition, whether the original

Fig. 3.—Leukoderma centrifugum acquisitum, beginning.

pigmented spot was a macule or a papule, the end result was always flat and level with the neighboring skin.

It may be argued that the central hyperpigmentations in leukoderma acquisitum centrifugum first come into being because melanin drains to the center, thus forming the moles, and that they then get involved by the "vitiligizing" process which erases them. We remember no report in which a medical observer claimed to have witnessed the first event of nevus formation. Therefore, it appears much more likely to us that nevi, lentigines and ephelides, which are so common to all, preexist or arise in the ordinary mysterious manner. Whenever they come into

being during life they tend to be permanent. They may, relatively rarely, disappear as a result of trauma or other uncertain reasons. In leukoderma acquisitum centrifugum they seem to be invaded by the same depigmenting process that concurrently affects the ordinary skin.

F. Parkes Weber [10] speculatively suggested that there may be a constant law embracing the phenomenon of depigmentation of this sort,

Fig. 4.—Same case as in figure 3, about eighteen months later. The darker portion in the center is erythema, not pigment.

as follows: Nevi predispose to circumferential depigmentation in an amount that is in direct proportion to their size. Stokes [7d] noted, and we have, too, that leukoderma acquisitum centrifugum is rather common

10. Weber, F. P.: A Law Regarding the Distribution of Pigmented (Leucodermic) Patches of Vitiligo, When They Are Superadded to Mole-Like Nevi, Brit. J. Child. Dis. **21**:202, 1924.

if sought for and that non-nevoid conditions, like the lesions of psoriasis, syphilis, Boeck's sarcoid and burns, are frequently surrounded by leukodermatous halos, and he therefore postulated that a common physicochemical inhibition of melanogenesis is at the basis of all such depigmentations. It seems to us that hyperpigmentations too are common around many dermatoses, including those mentioned by Stokes, none of which is commonly associated with problems of melanogenesis. Coupling our own observations with those of Weber and Stokes, we should say that there may be a law about pigmentary dyscrasia which may be formulated as follows: Any abnormality of the skin, congenital or acquired, perceptible or imperceptible, may become a locus of pigmentary disturbance in directions of excessive formation and concentration or of inadequate formation and dispersion. When the disturbance is in the direction of depigmentation, is imperceptible and is of point source and unknown cause on apparently normal skin, the clinical picture appears like that of banal vitiligo. When the predisposing disturbance, again in the direction of depigmentation, is an obvious lesion, then the clinical lesion may be leukoderma acquisitum centrifugum, with either a nevus or some other non-nevoid condition in the center. In the latter event, the word leukoderma is a most proper designation because the depigmentation is related to a known proximate cause. In reverse, the very same types of pigmentary disturbances may in other cases eventuate in chloasma-like changes. Indeed, vitiligo itself often seems to be accompanied with hyperpigmentation at its border, which observation supports a concept of melanogenesis as a mechanism of such delicate balance that the very same influence, or train of influences, seems to be able to direct it into the direction of either hypopigmentation or hyperpigmentation or of both simultaneously.

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IS LICHEN URTICATUS A FORM OF URTICARIA?

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SINCE the time of Bateman, who accurately described lichen urticatus, there has been controversy about the nature and pathogenesis of this disorder. In successive periods of dermatologic history new ideas have developed about this disease, reflecting the thoughts and subject matters current at the moment. Thus, at one period, foods and bowel disturbances were said to be causative, in harmony with the emphasis placed on intestinal putrefactions and toxemias. At present lichen urticatus is mostly thought of as a form of urticaria, in keeping with the keen awareness of the phenomena of allergy, and is so classified in the textbooks.

There has been so much written on lichen urticatus that there would seem to be little excuse for still further overtaxing what has been for the most part theoretic discussion. Only new evidence can be an adequate reason for reopening the subject. The recent development and use of the antihistaminic or antiallergic synthetic drugs may permit one to survey the problem from a fresh perspective. Toward that end the following several case histories are presented.

CASE HISTORIES

1. T. K. R., a Negro girl 4 years old, had a papular eruption mostly on the extensors of the extremities and a few lesions on the trunk and the face, which was diagnosed as lichen urticatus. The mother said it was present the previous two summers, too. After three weeks' use of diphenhydramine hydrochloride (elixir benadryl hydrochloride®) there was no improvement in the appearance of the lesions or apparently in the distress caused by the itching. There was marked betterment after three more weeks during which she was treated simultaneously with parathyroid injection U. S. P. and calcium lactate.

2. Z. P., a boy 5 years old, had an eruption which had been present in the same form the previous year. It was thought to be lichen urticatus. Four weeks' use of diphenhydramine hydrochloride failed to produce any evident improvement in the lesions, even though the child slept better and was more restful.

3. J. O. M., a woman 23 years old, had a pruritic papular eruption on the extensors of the arms and legs and scattered elements on the trunk. Some of the lesions were acuminate and there was severe pruritus. There was no grouping; nevertheless, she was given a course of sulfapyridine treatment on the pos-

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sibility that the eruption was dermatitis herpetiformis. There was no good effect from this therapy. She was then given 300 mg. of tripelennamine hydrochloride (pyribenzamine hydrochloride®) daily for ten days without visible or subjective improvement. Itching largely subsided and much of the eruption was gone after a month of the use of parathyroid injection U. S. P. and calcium lactate.

4. D. A., a white boy 8 years old, had a papular eruption mostly on the extensor surfaces of the extremities. A few lesions had puffy bases, and a frank wheal was present at one site. There was itching. He was thought to have lichen urticatus. The lesions remained largely unchanged after two weeks' use of diphenhydramine hydrochloride.

5. C. N., a Negro girl 6 years old, had an itching eruption which was believed to be lichen urticatus. Three weeks' use of diphenhydramine hydrochloride effected no apparent improvement in her cutaneous condition.

COMMENT

The greatest success attending the use of the antihistaminics has been in connection with urticaria. It is reasonable to assume that if lichen urticatus is a form of urticaria peculiar to children it should react somewhat similarly to therapeutic agents which benefit patients with urticaria. This small series of patients derived no benefit from the newer antiallergic agents in contrast with the happier results commonly seen in ordinary urticaria. This raises again doubts that lichen urticatus is primarily an urticarial process. Pruritus is common to both, but is present, too, where there is no suspicion of urticaria. The mere presence of wheals as one of the primary lesions should not be binding to a diagnosis of urticaria. It would seem tenuous to make their presence in urticaria pigmentosa a proper cause for including the latter among the urticarias even though its origin remains unknown. Wheals may at times be found in diverse entities, even as the initiating event of a folliculitis or a furuncle.

Walzer and Grolnick,[1] in an intensive study of lichen urticatus, employing modern methods of dealing with allergy, were unable to relate this disease to such a process. Pillsbury and Sternberg [2] said:

> . . . the theory that the nature of this disease is allergic has been commonly held, and most investigations have been concerned in the attempt to prove or disprove this contention. We believe that the evidence supporting such a view is extremely meager and that further study along the lines of allergy, unless some unique approach is devised, will probably be fruitless.

They reviewed the studies of various authors whose work had failed to resolve the beclouded pathogenesis of lichen urticatus and been incon-

1. Walzer, A., and Grolnick, M.: Relation of Papular Urticaria and Prurigo Mitis to Allergy, J. Allergy **5**:240, 1934.

2. Pillsbury, D. M., and Sternberg, T. H.: Lichen Urticatus (Papular Urticaria): Treatment with Parathyroid Extract; Theoretical Consideration of Etiology, Am. J. Dis. Child. **53**:1209 (May) 1937.

clusive toward allying this entity with urticaria. They attest to the efficacy of parathyroid extract used in conjunction with calcium in the former, compared with the mediocre results in the latter.

SUMMARY

The failure of the antihistaminic or antiallergic agents to influence lichen urticatus favorably is presented as further evidence to disprove any connection of this condition with urticaria.

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HEREDITARY ANHIDROTIC ECTODERMAL DYSPLASIA

A Clinical and Pathologic Study

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THE SYNDROME of hereditary anhidrotic ectodermal dysplasia, which has as its most prominent characteristics anhidrosis, hypodontia or anodontia and hypotrichosis, is one of a group of approximately 200 primarily cutaneous congenital abnormalities that are described in Cockayne's [1] extensive study of this subject. Of this group, the following five types of abnormalities were studied and described by one of us (B. Y. U.) in a Mayo Foundation thesis, "Congenital Ectodermal and Mesodermal Dysplasias; A Clinical and Pathological Study": (1) anhidrotic ectodermal dysplasia, (2) hidrotic ectodermal dysplasia, (3) erythroderma ichthysiforme, (4) pachyonychia congenita and (5) Ehlers-Danlos syndrome.

In our experience, each and all of these five types of abnormalities can well be called rare; less than 40 patients affected by them have been encountered at the Mayo Clinic in the past twenty-seven years. This paper will be confined to the study of the 4 patients affected by anhidrotic ectodermal dysplasia observed at the clinic since 1920.

Although, according to Darwin,[2] a description of this syndrome was given by Widderburn [3] in 1838, the term "congenital ectodermal defect" was not used until a case was reported by Christ [4] in 1913. Later observers also used this term until Weech [5] in 1929 designated the

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Abridgment of a thesis submitted by Dr. Upshaw to the Faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirements for the degree of Master of Science in Dermatology.

1. Cockayne, E. A.: Inherited Abnormalities of the Skin and Its Appendages, London, Humphrey Milford, 1933.

2. Darwin, C. R.: The Variation of Animals and Plants Under Domestication, New York, D. Appleton & Company, 1894, vol. 2, p. 319.

3. Widderburn, W.: Cited by Darwin.[2]

4. Christ, J.: Cited by Goeckermann.[14]

5. Weech, A. A.: Hereditary Ectodermal Dysplasia (Congenital Ectodermal Defect): A Report of Two Cases, Am. J. Dis. Child. **37**:766-790 (April) 1929.

group as "the anhidrotic type of hereditary ectodermal dysplasia," a term which we believe to be most descriptive, because it emphasizes both the cause and the most important clinical symptom of the syndrome, inability to sweat.

According to recent reviews of the literature, including those of Kaalund-Jørgensen and Christensen [6] and Helweg-Larsen and Ludvigsen,[7] approximately 80 cases of the complete anhidrotic syndrome had been reported prior to 1947. The syndrome has a wide racial distribution, reports having been made from many of the countries of North America, Europe and Asia.

Although this triad of symptoms, namely, hypodontia, hypotrichosis and anhidrosis, has been observed many times during the past hundred years, the literature we have read includes no observation of universal anhidrosis that occurred alone. This is unusual when one considers that universal aplasia of other single components of the skin, such as hair or pigment to produce alopecia totalis or albinism, respectively, is not infrequent.

CLINICAL CHARACTERISTICS

The patients affected by the anhidrotic type of ectodermal dysplasia resemble each other to a remarkable degree because of their unusual facies, which is somewhat suggestive of congenital syphilis and is characterized by high, wide brows, prominent frontal bosses, depressed nasal root and bridge, thick lips with radiating furrows about the mouth, and satyr-like ears.

The skin of these patients is unusually soft, thin and feminine. Total or almost total lack of sweat gland function under all environmental conditions is the most important physiologic feature; sebaceous gland activity is usually diminished to a variable degree, but apocrine function may be normal. The mammary glands may be normal or, as described by Clarke and McCance [8] as well as others, they may be deficient or absent.

A delayed appearance and a marked deficiency of development of scalp hair, eyebrows and eyelashes and lanugo are frequent, but there is often little uniformity in the degree of development of hair beginning at or after puberty, the beard being more frequently normal, while axillary and pubic hair is either scanty or absent. Scalp hair is usually stiff, fine and short.

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6. Kaalund-Jørgensen, O., and Christensen, J. F.: Congenital Ectodermal Dysplasia of the Anidrotic Type, Acta dermat.-venereol. **22**:1-23 (Feb.) 1941.

7. Helweg-Larsen, H. F., and Ludvigsen, K.: Congenital Familial Anhidrosis and Neurolabyrinthitis, Acta dermat.-venereol. **26**:489-505 (May) 1946.

8. Clarke, R. E., and McCance, R. A.: Familial Sex-Linked Ectodermal Dysplasia with Incomplete Forms, Arch. Dis. Childhood **9**:39-44 (Feb.) 1934.

The nails of anhidrotic patients are often normal but may exhibit slow growth, longitudinal furrows or slight atrophy.

Delayed dentition and hypodontia regularly occur in these patients. There is not only a deficiency in number of both deciduous and permanent teeth, but the teeth present are usually abnormally placed and their normal anatomic markings are frequently absent. Complete anodontia accompanying anhidrosis has been reported by several authors, including Guilford [9] and Thoma and Allen [10]; however, complete roentgenologic proof of anodontia is not given by all of these authors. The erupted teeth, particularly the incisors and bicuspids, are often widely separated, peg shaped or conical, darkly pigmented and fragile. The development of the maxilla and mandible is in proportion to the number of erupted teeth and usually depends on the amount of resulting mastication made possible, but bony development may be normal in patients with complete anodontia.

We have found in the literature no reference to congenital cataracts in connection with this syndrome, although the lens is of ectodermal origin and such an anomaly might be expected. Many authors, however, do report deficient function of the lacrimal glands. Along with their nasal deformity, many of the patients have chronic rhinitis, often accompanied by nasal discharge of a foul-smelling fluid and diminution of the olfactory sense. The glands of the mucous membrane of the nose and throat have repeatedly been found to be deficient in secretory function, producing a decrease in sense of taste, dysphagia, hoarseness and intermittent complete aphonia.

Although their anomalies are of ectodermal origin, only rarely have definite defects of the nervous system been found in typical anhidrotic patients. It is true, however, that numerous anhidrotic patients have been mentally inferior, but it is commonly believed that the same genetic mutation is not responsible for both of these anomalies.

In addition to the aforementioned clinical characteristics which are diagnostic of ectodermal dysplasia of the anhidrotic type, other abnormalities occasionally and probably coincidentally have been associated with it, including skeletal anomalies and dysfunction of the adrenal medulla and the pituitary gland.

HISTOPATHOLOGIC CHARACTERISTICS

The majority of observers who have studied excised epidermis from anhidrotic patients have reported either a total absence of sweat glands or the presence of rudimentary fragments of nonfunctioning glands and

9. Guilford, S. H.: A Dental Anomaly, Dent. Cosmos **25**:113-118 (March) 1883.

10. Thoma, K. H., and Allen, F. W.: Anodontia in Ectodermal Dysplasia, [case 38], Am. J. Orthodontics **26**:503-507 (May) 1940.

ducts. There is usually a similar marked deficiency of pilosebaceous structures, but this anomaly is more variable in degree. The epidermis is usually thinner than normal, and there is likewise a reduction in width of the cutis, but collagen and elastic fibers and blood vessels are normal in appearance. Sunderman [11] reported normal apocrine glands but absent eccrine glands in his biopsy material from the axillas of 2 patients. In the anhidrotic patients of Helweg-Larsen and Ludvigsen, as microscopic sections revealed, the number of sweat glands was much reduced, while the glands which were present were hypertrophic.

Biopsies of the thin, dry mucous membranes of the mouth and upper respiratory passages have revealed hypoplastic changes in the epithelium as well as in the underlying glands, corresponding with the clinical appearance of these structures.

PHYSIOLOGIC CHARACTERISTICS

It must be pointed out that evidence of complete anhidrosis based on single or multiple biopsies is incomplete, even though the sites include the palm or the sole and serial sections reveal no sweat glands. Specific physiologic studies of temperature and sweat are necessary for proof of complete anhidrosis. At ordinary environmental temperatures and at rest, when the sweat glands are not needed to dissipate heat, the amount of insensible perspiration given off by cutaneous transudation of water is quite sufficient to maintain a normal temperature of the body in anhidrotic as well as in normal patients. However, at high environmental temperatures, when a temperature of 98.6 F. can be maintained in a normal person by the function of sweat glands, the temperature of the anhidrotic patient rises rapidly to extremes of 102 to 104 F., and the pulse and respiratory rates increase proportionately in a vain attempt to dissipate heat.

Although the amount of perspiration of anhidrotic patients is inadequate to maintain a normal body temperature at high environmental temperatures, Felsher [12] has observed that some few sweat glands may be present and functioning in these patients. By applying a starch-iodine mixture to the skin of his patients, he saw a few small areas where sweat was being produced when the skin was exposed to heat. He therefore suggested that the term "hypohidrosis" rather than "anhidrosis" be applied to this group of patients.

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11. Sunderman, F. W.: Persons Lacking Sweat Glands; Hereditary Ectodermal Dysplasia of the Anhidrotic Type, Arch. Int. Med. **67**:846-854 (April) 1941.

12. Felsher, Z.: Hereditary Ectodermal Dysplasia: Report of a Case, with Experimental Study, Arch. Dermat. & Syph. **49**:410-414 (June) 1944.

ETIOLOGIC FACTORS

Because of the facial similarity of anhidrotic patients and patients with congenital syphilis, clinicians formerly believed the cause of the anhidrotic syndrome to be congenital syphilis. Still others explained the syndrome on the basis of endocrine dysfunction, but it now appears that all etiologic concepts other than those of heredity have been disproved.

The first anhidrotic patients described by Widderburn,[3] were 10 male members of a Hindu family, and although many patients have been reported who gave absolutely no family history of abnormalities, the majority of patients have ancestors with similar anomalies. When the full syndrome is not found in other members of the family, several of them will often have one or more of the component anomalies, except anhidrosis, that comprise typical anhidrotic dysplasia.

The exact mode of genetic transmission of the full anhidrotic syndrome has been the subject of controversy since the first cases were encountered. It was noted early that males are affected much oftener than females; of the first 60 patients, only 8 were females, according to Kaalund-Jørgensen and Christensen,[6] who also listed 7 instances in which the typical syndrome occurred in silblings. Wechselmann concluded from his studies that the defect was transmitted as a sex-linked characteristic, in the same manner as hemophilia, by an unaffected female conductor to affected male members of the family.[13] Goeckermann,[14] in a report on a female patient, pointed out that the syndrome was not always recessive in females. As more and more cases were reported, it became obvious that the mode of genetic transmission is not identical in all patients. Geneticists now emphasize that one peculiarity of human genetics is that the same abnormal character may be sex linked, dominant or recessive in different families, and there are now reports in the literature of the genetic transmission of the complete anhidrotic syndrome by each of these modes of inheritance.

PATHOGENIC FEATURES

Although the time and mode of production of the anhidrotic syndrome constitute a subject of much speculation, little detailed information is known.

The relative order of normal embryologic development of the dermal appendages which are involved in the syndrome is as follows: 1. The hair develops from downgrowths of the epidermis which appear

13. Wechselmann, W., and Loewy, A.: Untersuchungen an drei blutsverwandten Personen mit ektodermalen Hemmungsbildungen, besonders des Hautdrüsensystems, Berl. klin. Wchnschr. **2**:1369-1373 (July) 1911.

14. Goeckermann, W. H.: Congenital Ectodermal Defect, with Report of a Case, Arch. Dermat. & Syph. **1**:396-412 (April) 1920.

between the second and third months of gestation, first in the region of the eyebrows and forehead. Between the fourth and fifth months, rudiments of hair begin to grow from these follicles. 2. The earliest evidence of nails appears during the third month, but formation of the nail matrix occurs later, at the fifth month of gestation. 3. Sebaceous glands originate as lateral outgrowths of the hair follicles during the fifth month. 4. Sweat gland anlages appear first on the tips of the phalanges between the fourth and fifth months, and on the proximal portion of the extremities and the trunk by the end of the sixth month; development of these glands is almost complete before birth.

The remainder of the embryologic data concerned in the syndrome is as follows: 1. The teeth have a double origin; the enamel organ develops from the ectoderm, and the dentine, pulp and cement develop from the mesoderm. By the seventh week of gestation, the dental lamina of the ectoderm shows circumscribed thickenings where the future teeth are to be formed. At eight weeks, the underlying mesodermal cells begin their growth and differentiation to form the future mesodermal portion of the teeth. 2. The milk ridge appears in the embryo between the fifth and sixth weeks of intrauterine life, and from it, in the pectoral region, papilla-like anlages of the mammary glands develop early in the fifth month. 3. The neural tube, from which the entire nervous system, the adrenal medulla and a portion of the pituitary gland develop, is completely closed and separated from the overlying epidermis by the beginning of the second month of gestation.

Since few reports of definite abnormalities of the nervous system in anhirotic patients have been recorded, it seems probable that the pathologic process resulting from the defective gene or genes begins to affect the development of the epidermal structures during the second fetal month. However, the lens, although undergoing separation from the ectoderm during this time, enjoys, to our knowledge, complete immunity. Although the mechanism of genetic transmission of these defects is fairly well understood, at this time the method by which certain structures appear to be haphazardly affected by these genes is not at all clear.

Most observers have agreed that the pathologic process is probably an aplasia and not an atrophy because degenerative changes in ectodermal structures rarely have been seen in tissue sections; the structures instead appear never to have begun or just barely to have begun to differentiate.

DIFFERENTIAL DIAGNOSIS

There are four diseases which usually have enough symptoms in common with the anhidrotic syndrome to be considered in its differential diagnosis. The accompanying table is a brief summary of their essential similarities and differences.

TREATMENT

Treatment of anhidrotic patients is largely symptomatic because of the fundamentally aplastic lesions of the syndrome, but certain therapeutic principles should be emphasized to the patient or to the parents of an anhidrotic infant. The limiting effect of the anhidrosis on the mode of living must be well understood; the genetic transmission of

Differential Diagnosis of the Anhidrotic Syndrome

	Anhidrotic Ectodermal Dysplasia	Hidrotic Ectodermal Dysplasia	Werner's Syndrome	Rothmund's Syndrome	Progeria, Hutchinson-Guilford Type
Genetic transmission	Dominant, recessive or sex linked	Usually dominant	Usually recessive	Usually recessive	Not proved
Age at onset of symptoms	First year	First year	20 to 30 yr.	1 to 5 yr.	1 to 5 yr.
Facies	Inverted pyramid, frontal bosses, depressed nasal bridge	Not characteristic	Senile changes, beaked nose, small mouth and chin	Not characteristic	Frontal bosses, beaked nose, retracted chin
Skin	Dry, soft, feminine	Hyperkeratoses of palms and soles, oriental pigmentation	Epidermal, subcutaneous and muscular atrophy of distal extremities with trophic ulcers	Atrophy, brownish pigmentation, telangiectases	Atrophy of epidermis, subcutaneous tissue and muscle
1. Sebaceous glands	Markedly decreased	Slightly decreased	Normal	Normal	Normal
2. Sweat glands	Decreased or absent	Normal	Normal	Normal	Normal
3. Mammary glands	May be absent	Normal	Normal	Normal	Normal
4. Hair	Generalized alopecia, usually subtotal	Generalized alopecia, always subtotal; fragile hair	Canities and premature alopecia	Usually normal	Premature alopecia
5. Nails	Normal	Short, thick, elevated tip	Normal	Normal	Short, fragile
Teeth	Decreased or absent, also deformed	Normal	Normal	Normal	Irregular development
Eyes	Normal	Normal	Juvenile cataracts	Juvenile cataracts	Normal
Systemic abnormalities					
1. Cardiovascular	Distinct intolerance of heat	None	Early arteriosclerosis	None	Early arteriosclerosis
2. Skeletal	None	Usually none	Short stature, osteoporosis	Occasionally short stature and small hands and feet	Dwarfed trunk and extremities, joint deformity
3. Endocrine	Usually none	Pituitary and suprarenal deficiency (?)	Hypogonadism, diabetes	Occasional hypogonadism	Hypogonadism
Prognosis for life	About average	Average	Death before age of 40 yr.	Average	Death before age, of 20 yr.

the defect should also be explained, and no assurances should be given to the parents of an anhidrotic child that future children will be normal.

Artificial dentures should be fitted early in anhidrotic children with hypodontia to facilitate the development of the mandible and the maxilla. Plastic repair of the saddle nose deformity will likewise do much to improve the patient's facies and general morale. Mortimer, Wright

and Collip [15] have reported favorably on the results of using estrogenic substances incorporated in a nasal spray in treating anhidrotic patients for chronic rhinitis or ozena. In view of the normal longevity enjoyed by the patients affected by anhidrosis, the aforementioned measures, although not curative, are certainly important in making life more pleasant.

REPORT OF CASES

Case 1.—A white boy, 7 weeks of age, was brought to the clinic during warm weather because of intermittent fever that had been present since 2 weeks of age. The baby weighed 6 pounds 8 ounces (2,950 Gm.) at birth and appeared normal. Except for the fever of unknown origin, there were no symptoms of illness. The family history revealed numerous congenital abnormalities, none of which could be related to the anhidrotic syndrome. The child had no siblings, and there was no history of intermarriage of relatives.

In the physical examination, the only abnormal findings were absence of eyelashes and eyebrows, and very dry skin over the entire body. In the hospital it was noted that the child never perspired and that his temperature increased directly in relationship to the external environment. After fifteen minutes under a heat cradle, the rectal temperature rose from normal to 100 F.; increases in temperature were noted after each meal. No teeth were visible on roentgenologic examination. Routine laboratory investigations revealed moderately severe hyperchromic anemia. A biopsy specimen was removed from the sole of the left foot, and the multiple sections studied revealed complete absence of sweat glands and ducts, the epidermis and cutis being normal in other respects.

Four years later the mother said that no sweating had ever been noted and that by living in the basement during hot weather he had been quite comfortable and able to maintain a normal temperature. At 18 months of age he had two pointed teeth which were still present and strong at the age of 4 years. His scalp hair was white, sparse and slow growing; he had normal eyelashes but no eyebrows. The mucous membrane of his nose was dry and crusted, and he had had several severe laryngeal infections, one of which had necessitated tracheotomy. His mental development was thought to be normal.

Case 2.—A white boy, 2 years old, was brought to the clinic from a warm climate by his paternal grandmother who said that since shortly after the birth of the child it had been noted that his temperature rose when he was placed in a warm environment. The patient weighed 5 pounds (2,270 Gm.) at birth, six hours after this event his temperature reached 106 F. Fever continued almost constantly during the first summer, and no cause could be found. When cool weather began, the child's temperature returned to normal, but it began to rise on the first warm day of the following year. His pupils had been dilated since birth; they did not become smaller in the bright sunshine, and photophobia was obvious. The child had had eczema of scalp, hands and feet since 6 months of age, and this was much more severe in warm weather. When the teeth appeared, they were brown and fragile and soon became carious. At 1 year of age, eight teeth had developed. The nails had been normal until 1 year of age, when they became rough and scaly. The child sat alone at 8 months and walked at 18 months.

15. Mortimer, H.; Wright, R. P., and Collip, J. B.: Atrophic Rhinitis: The Constitutional Factor; And the Treatment with Œstrogenic Hormones, Canad. M. A. J. **37**:445-456 (Nov.) 1937.

The family history revealed no congenital abnormalities, but the patient's paternal great-grandfather and maternal great-grandmother were brother and sister. The patient's one sibling, a sister of 8 months of age, was said to have the same abnormalities of eye and temperature as were noted in the patient.

On physical examination, the child appeared to be well developed and nourished. The facies and the hair distribution were not unusual, but the skin was dry, and no sweating was noted. No eczematoid lesions were present, and the teeth and nails were as described. The pupils were equal and semidilated and did not react to light. Pupillary dilatation did not occur after local instillation of homatropine hydrobromide nor constriction after instillation of physostigmine salicylate or a pilocarpine salt. There were pupillary remnants in both eyes, and the fundi were normal. The remainder of the physical examination revealed no significant findings, and the laboratory examinations gave essentially normal results.

Further observations of the child revealed that his temperature increased after eating and with crying but could be easily controlled by sponging him with cool water. After administration of pilocarpine there was flushing of the face, neck and breast down to a sharp line at the nipple level, but no sweating occurred. Biopsy of the skin was not performed.

When the patient was 5 years old, the grandmother stated that he still was intolerant to heat, that there was no change in his pupils and that his deciduous teeth were black stumps except his six year molars, which appeared normal. No sweating had ever been noted in the patient's younger sister, and although she had a complete set of deciduous teeth, they were darkly pigmented and sensitive to sweets, heat and cold.

CASE 3.—A white man, 26 years of age, unmarried, working as lifeguard, first came to the clinic during warm weather because of lack of perspiration, scanty growth of hair and abnormal dentition. These abnormalities had been present since birth, and the father stated that the patient's abnormalities were familial in character, originating many generations ago in the patient's mother's family, always appearing in one male of each generation and completely absent in the women of the family as far as he knew.

Physical examination revealed a well nourished man of average adult size; the ears were protuberant; the root of the nose was depressed and the septum was irregular, but there was no widening or saddle nose deformity. The skin was soft and dry. On the scalp there was only a sparse peripheral fringe of hair; there was little lanugo over the trunk and extremities, and only a few small tufts of hair in the pubic and axillary regions. Two teeth were present, one of which was a small tag. There was moderate atrophy of the mucosa of the nasopharynx, pharynx and larynx, but the voice was normal.

Biopsy and other laboratory procedures were not performed until the patient was admitted to the clinic a second time, five years later. Then it was found that after strenuous physical exercise the rectal temperature increased 2 degrees F. His basal metabolic rate was normal on two occasions, but after strenuous exercise it increased to + 40 per cent, while the metabolic rate of the normal control person increased to + 20 per cent. After his second admission, histologic examinations were made of skin tissues removed from the right axilla and the lower right part of the thorax; in the serial sections no trace of hair follicles, sebaceous glands, sweat glands or apocrine glands could be found.

CASE 4.—A 23 year old unmarried farmer came to the clinic because of weakness of the arms and legs and loss of consciousness, usually occurring just preceding a meal or several hours afterward. These symptoms began suddenly during one hot day of the preceding summer and continued to recur during cooler weather. He

had had no convulsions at any time and had noted no aura prior to loss of consciousness. The patient had been told elsewhere that he had congenital absence of the sweat glands, and he had noted heat intolerance all his life. He did not relate the onset of weakness or unconsciousness to becoming overheated or to hyperventilation. The family history concerning congenital defects did not disclose anything that was significant. Two brothers had died in early infancy of unknown causes.

Physical examination revealed a young man of average height and weight (fig. 1). His forehead was extremely high; his small eyes were widely separated. His nose was wide, the bridge being almost flat. The lips were thick and muscular; there was total anodontia, and the mandible and the maxilla were underdeveloped, producing a "dish face" deformity.

Fig. 1 (case 4).—(*a*) Anterior and (*b*) profile views. This is the characteristic concave facies of hereditary anhidrotic ectodermal dysplasia. Note the sparse growth of hair on the scalp and the normal amount of lanugo in the shoulders.

No activity of sebaceous or sweat glands was noted anywhere on the skin, which was excessively dry. A diffuse symmetric keratoderma covered the palms; the soles and the nails were normal.

There was considerable alopecia of the scalp, most prominent over the vertex. The scalp hair present was stiff and dry, and only a few hairs were present anteriorly. Eyelashes and eyebrows were absent, and only a few sprigs of hair were present in the axillary and pubic regions. The beard was normal, as was the lanugo on the thorax and the shoulders, but lanugo was absent elsewhere.

The mammary glands were absent, and there was no pigmentation to mark their usual site. No teeth were present. The eyes and the lacrimal glands were normal.

The thin septum of the nose contained no cartilage, and the mucous membrane was atrophic. The senses of smell and taste were intact.

Routine examinations of blood and urine gave normal results, as did determinations of the basal metabolic rate and the fasting blood sugar, calcium and potassium. Roentgenograms of the skull and the thorax did not disclose any abnormality, and the electroencephalogram revealed mild generalized dysrhythmic activity without localizing signs.

Under an electric baker the patient's oral temperature rose from 98.2 to 100.4 F. in fifteen minutes. After an injection of 5 mg. of furfuryl trimethylammonium iodide (furmethide®) flushing and weakness were noted, but no sweat, and no tremor or unconsciousness developed.

Fig. 2 (case 4).—Section of a biopsy specimen removed from the palm. It shows complete absence of sweat glands and ducts (hematoxylin and eosin; × 25).

Biopsy specimens were removed from the skin of the right forearm, right arm and palm of the right hand; in the serial sections no trace of coil glands or ducts was visible (fig. 2).

Over a period of several months, three plastic procedures employing cartilage implants succeeded in forming an elevated nasal bridge, so that the patient's appearance was improved. Diphenylhydantoin sodium (dilantin sodium®) was prescribed in an attempt to control the episodes of weakness and unconsciousness; the patient was advised to restrict his strenuous farming activities during hot weather.

COMMENT ON CASES

As in case 1, it is not uncommon for anhidrotic infants who have increased temperature shortly after birth to be extensively and vainly studied for evidence of infections. Seldom is the correct diagnosis made in the first few weeks of life, and so the child is not subjected to varied and multiple therapeutic methods. In contrast to those in adults, the obvious clues to the correct diagnosis, namely, anodontia and hypotrichosis, are normal findings at this age. The typical anhidrotic facies, although observed as early as 1 year of age, is not conspicuous in the first few weeks of life, and in the absence of a history of similar hereditary anomalies, unless the family or the physician notes that the rise of temperature is correlated with the external environment, the diagnosis may be delayed until lack of teeth and hair is noted in the second six months of life. In case 1, since the patient fortunately was examined in August, the correlation of high body temperature and high external environmental temperature was noted; when, in further study of this phenomenon, the patient was exposed to a heat cradle and no sweating was produced at an abnormally high body temperature, only the biopsy showing absence of sweat glands on the sole of the foot was needed to confirm the diagnosis. The absence of teeth on roentgenologic examination of the mandible and the maxilla was further evidence of the existence of the complete anhidrotic syndrome. In the child's subsequent development, the repeated infections of the upper part of the respiratory tract illustrated one of the common complications resulting from the abnormal formation of the mucous membrane.

Of particular interest in case 2 were the pupillary abnormalities and the mode of inheritance of the syndrome, the latter factor being unknown in case 1. The inability of the pupils to respond to any type of physical or chemical stimuli was thought to be due to defective innervation, though the site of this abnormality of the nervous system could not be determined in absence of other neurologic signs or symptoms. This anomaly appears to be one of the rare examples of abnormalities of the nervous system that accompany the complete anhidrotic syndrome.

In case 2 all of the patient's manifestations of anomaly were said to be duplicated in his only sibling, a sister, 8 months old. When one remembers that the children's parents had common great-grandparents each of which may have carried the anomaly as a recessive genetic character, it is easy to understand why marriage between their descendants resulted in defective children. These are obviously homozygous for the causative gene, and this recessive gene was concealed

until the consanguineous marriage by a dominant normal gene, that was absent in these two children. Progeny of heterozygous parents would be expected to show a 1:3 ratio of defective to normal children, but this ratio often becomes apparent only in large families or in long family pedigrees.

In case 3 a sex-linked recessive gene was responsible for the defect, according to the father of the patient, since only one male of each generation was affected. In contrast, in case 4 we were unable to trace the anomaly at all. One must conclude that the condition in this case represents a mutation, and the patient's progeny should be followed with interest to note the genetic transmission of the defect.

The facies of the 2 adult anhidrotic patients (cases 3 and 4) is characteristic of the entire group. Although in case 3 the patient did not have a saddle nose such as that described in case 4 and frequently described in the literature, the root of his nasal bridge was sharply depressed. Both patients presented decided hypotrichosis, complete anhidrosis and hypodontia or anodontia to complete the full anhidrotic syndrome.

The anhidrotic syndrome had limited the heat regulation in case 3 to such an extent that the patient's employment as a lifeguard was one of the few types of work which he could carry on in the summertime. In case 4 the patient's activity was voluntarily restricted, but he was able to continue farm work in a northern state during almost the entire year. We were unable to relate the cause of his periods of weakness and unconsciousness directly to increase in body temperature, but it was felt that a subthreshold cerebral dysrhythmia was made manifest either by the rise in temperature or by the hyperventilation or by both factors.

These patients (cases 1, 3 and 4) were all of interest from the standpoint of histopathologic study. In none of their biopsy specimens were we able to find sweat glands or sebaceous glands. In case 3 the apocrine glands were also absent from the axillary skin examined. This is in contrast to the reports of Sunderman,[11] whose patients had apocrine glands but no eccrine glands, as shown by microscopic examination. The apocrine glands develop from anlages of hair follicles instead of directly from the epidermis as do sweat glands, in the embryo, and do not become active until near puberty. The presence of highly developed apocrine glands and the sparse growth of hair, on one hand, and the absence of sweat glands, on the other, illustrate the complete suppression of the sweat gland formation and the incomplete suppression of pilosebaceous structures which are peculiarly characteristic of the anhidrotic syndrome.

SUMMARY

The clinical characteristics of hereditary anhidrotic ectodermal dysplasia are described, emphasis being placed on the diagnostic triad of symptoms: anhidrosis, hypodontia and hypotrichosis. The most important dermatopathologic abnormality, complete or almost complete lack of formation of sweat glands, is pointed out, along with its effect on the patient's heat-regulating mechanism. The several modes of hereditary transmission of the syndrome, as well as the embryologic development of the anomalies, are discussed. The essential similarities and differences between the anhidrotic syndrome and hidrotic ectodermal dysplasia, Werner's syndrome, Rothmund's syndrome and the Hutchinson-Guilford type of progeria are pointed out in tabular form. Four patients who were affected by the anhidrotic syndrome, 2 of them infants, are reported on in detail. The report includes physiologic investigations of sweating, histopathologic examinations of epidermal tissue and inquiries regarding the genetic transmission of the anhidrotic syndrome in each patient's family group. There is need that the syndrome be recognized promptly, particularly in an infant, so that the child may not be exposed to a warm environment and, as his development progresses, may have the benefit of dental prostheses early enough to prevent some of the facial abnormality which results from uncorrected hypodontia or anodontia.

FLEA PROBLEM IN CALIFORNIA

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THE TESTIMONY of present day dermatologists is that fleas are more of a nuisance in California, particularly in the San Francisco Bay area, than elsewhere in the United States.

HISTORICAL ACCOUNTS

In the vast literature relating to exploration, settlement and life in California the references to misery caused by fleas encountered by the authors are related entirely to experiences in Northern California.

Padre Juan Crespi,[1] the diarist of the Portola expedition of 1769, wrote that when the expedition visited an Indian village on Purisima Creek in San Mateo County they found the grass huts so infested with fleas that they called it "Village of the Fleas."

In 1786 the Frenchman La Perouse [1] had this to say about the fleas in Carmel and Monterey: "Under the influence of the Missionaries the Indians had refused to make the slightest change in the construction of their huts because they could set fire to their huts when the fleas became a pest and could rebuild them in two hours."

Another Frenchman, Duhart-Lilly (1827-1828) [1] visited a ranch in the San Bruno Valley. Concerning the sleeping arrangements he wrote, "We were compelled to stow ourselves away, guests, husband, wife and children all together, upon a great feather bed, where devoured by fleas . . . we passed quite an uncomfortable night."

In the period of transition from Mexican sovereignty to that of the United States, the American Edwin Bryant, in his outstanding book, "What I saw in California," [1] wrote bitterly and in detail of the infestation of the rancheros and old missions with fleas. He spoke of the missions as "nurseries of fleas." "If any sinning soul ever suffered the punishment of purgatory . . . these torments were endured by myself last night," he said. After a sleepless night there was no square inch of his body that was free from flea bites.

Read at the Sixty-Eighth Annual Meeting of the American Dermatological Association, Inc., San Diego, Calif., April 28, 1948.

1. The Flea in California History and Literature, California Hist. Soc. Quart. **15**:329 (Dec.) 1936.

The Rev. Walter Calton (1850), in his book "Three Years in California,"[2] described the continued annoyance caused by fleas to strangers and observed that the native was not troubled by them.

Carl Meyer (1855), under the title "Nach dem Sacramento,"[1] described a night spent on a ranch near Salinas. He labeled his experience "flea fever."

> Hardly has one gone to bed when a band of these small devilish fleas pursue their bloody maneuvers on one's sensitive skin, driving away sleep and tormenting a man to madness. One breaks out in a torturing sweat, and because of the frequent throwing off of the covers one runs a risk of contracting a fever.

He mentioned that even in mixed company the subject of fleas was often the topic of conversation and that the gesture of scratching was not frowned on.

Ernest de Massey wrote in his "Frenchman in the Gold Rush"[1] of his experiences in San Francisco.

> There were millions of fleas in the sand near my shack. . . . On the floor was a layer of sand, the camping ground of a colony of fleas, which hopped around looking for a chance to make a good meal off me . . . [and] by the time I was ready to retire my legs were literally covered with these blood thirsty insects.

Frank Marryat, an Englishman, in his "Mountains and Molehills"[1] described his experiences in Santa Rosa, Calif. On being shown his quarters he was "immediately attacked by the fleas with a vigor which was perfectly astonishing." He had been a wide traveler and had "been tortured by sand fleas in the Eastern Archipelago and by all kinds of mosquitoes from Malta to Acapulco," including the "famous 'tiger' breed against which there is no recourse but flight." "But," he said, "I would have preferred any of those annoyances to the attack of those Santa Rosa fleas." "The place was alive with them and they, raised in the rough school of the wild bullocks' hide, boldly faced as they attacked us." He stated that, on his mentioning the fact, his host predicted that he would get used to the fleas—that he [the host] and his family never gave them a thought. Thus the early settlers in California had observed that one became immunized against the effect of flea bites by being bitten by the fleas.

In the San Francisco *Argonaut* of Dec. 8, 1878,[1] there appeared a humorous article with the title "The Flea—A Short Discourse on a Lively Subject." The author gave an account of his experiences with fleas encountered near what is now Redwood City. At that time it was believed that fleas lived in the soil. The author chose a site for his

2. Calton, W.: Three Years in California, New York, A. S. Barnes & Co., 1850, p. 70.

home near a ranch called "Rancho de las Pulgas," where the beauty of the country impressed him. He observed that potatoes planted in the soil had to be removed with a crowbar because of the hardness of the soil and that the potatoes when dug were as "flat as pancakes." He thought that fleas could not live in such soil. On calling on the owner of the ranch he noticed a stick ladder leading from the floor to a high attic under the roof, where the household slept. The significance of such an arrangement became apparent when, on being severely bitten about his ankles, he looked down and saw that his white socks were covered with "pepper in motion." The home owner then told him that his routine for sleep was to remove all his clothes, leave them on the floor and scurry up the ladder, leaving only the clothes for food for the fleas. Subsequently the author learned that "Rancho de las Pulgas" signified "The Farm of the Fleas."

ENTOMOLOGY OF FLEAS [3]

The three species of fleas most commonly attacking man in the United States are the human flea (Pulex irritans), the cat flea (Ctenocephalides felis) and the dog flea (Ctenocephalides canis). In the eastern part of the United States the dog flea is the dominant pest. In the South and West the human flea is. In California the human flea and the cat flea are equally dominant (fig. 1).

The eggs of all these species hatch in the nests or resting places of their hosts, rarely on their bodies, or else in dust or organic debris in buildings. Following the life cycle of any insect, from egg to larva to pupa within the cocoon, the adult flea emerges in about two weeks. Newly emerged adults are usually very fat and can live for several months, under favorable conditions, without feeding. The female flea must have a blood meal before her eggs can be fertilized. Most fleas visit the body of the host for relatively short periods for the purpose of obtaining a blood meal.

On smooth surfaces, fleas progress by means of short jumps. They may sham death when disturbed. They usually leave a dead host as soon as the body becomes cold. They like warmth and moisture but shun daylight. It has been determined that some adult fleas can jump vertically nearly 8 inches (20 cm.) and horizontally 13 inches (33 cm.). Fleas prefer hosts with nests.

The life cycles of fleas are decidedly influenced by meteorologic factors. Low atmospheric humidities in particular are inimical to them.

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3. The data and some of the phrasing on entomology were given to me by Morris A. Stewart, Ph. D., Professor of Parasitology, the Division of Entomology and Parasitology, University of California, Berkeley, Calif.

The length of time required for each developmental stage is very largely determined by the prevailing temperature and humidity. Adult fleas may remain alive for more than five years if the environmental conditions are favorable; such extremely long adult life is, of course, very exceptional; usually the flea lives only a few weeks or months. Because of the lack of extremes in temperature and the relatively high humidity, in coastal California fleas breed the year around. They are found in homes where cats and dogs are kept as pets, particularly in the carpets and cracks of floors and corners of rooms. They are found in basements where the animals sleep at night. They are particularly apt to be found in large numbers in such basements from which the usual animal

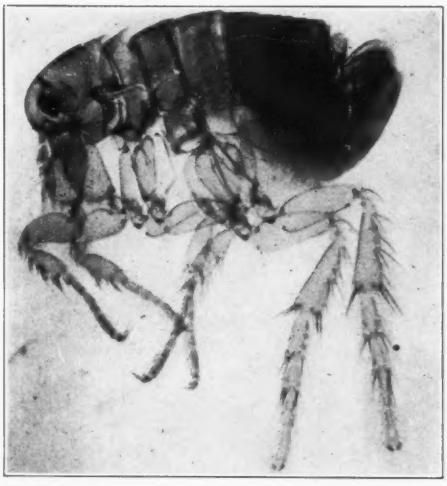

Fig. 1.—Cat flea. (Courtesy of Morris A. Stewart, Ph.D., Department of Entomology, University of California, Berkeley, Calif.)

and human tenants have been absent for a period. I know of persons who have walked into their own basements after a summer vacation, to have their white trousers literally covered with very hungry fleas in a few seconds. Fleas also frequent outhouses, sand piles and lawns and such places as theaters. I know of people who are bitten by fleas whenever they attend any theater.

CLINICAL PICTURE

The typical clinical picture of reaction to flea bites is a grouped, itching, urticarial, papular eruption, some papules of which contain a central red punctum (fig. 2). These lesions are most often found along the

waist line and on the hips and shoulder area, where the clothing fits snugly (fig. 3). They may, however, be on any part of the body. Grouping is one of the most characteristic features. The flea is not content to bite in one area and remain attached at that point until its hunger is satisfied. Rather, it moves along, biting in an irregular but grouped

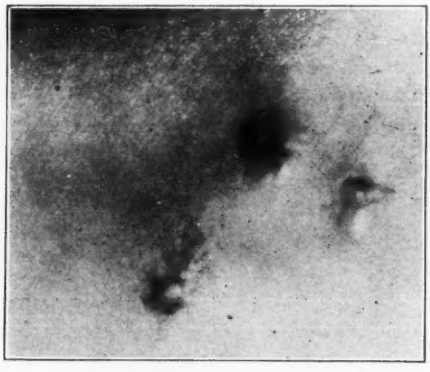

Fig. 2.—Individual flea bites, showing papules, central puncta and vesiculation.

Fig. 3.—Flea bites in typical round the waist distribution. (Courtesy of Rees B. Rees, M.D., San Francisco.)

pattern. In addition, there may be more scattered or satellite lesions present. In some cases a generalized papular urticaria is seen (fig. 4). It is believed that many of such papules are allergic reactions to one or a few bites and are an expression of sensitivity to the antigen injected

by the flea as he bites. Or, to put it in other words, a flea may bite, and as a result there may be urticarial lesions at sites remote from the bitten areas.

The history is equally important. If the sufferer is adult, he is usually new to the Bay region. He has been subject to flea bites for only a sufficient period of time to become sensitized by the antigen. Most patients suffering from flea bites are children, newly born or newly arrived in the Bay area.

Other types of eruptions which are seen more rarely are secondary bacterial infections and other complications of scratching. Recurrent

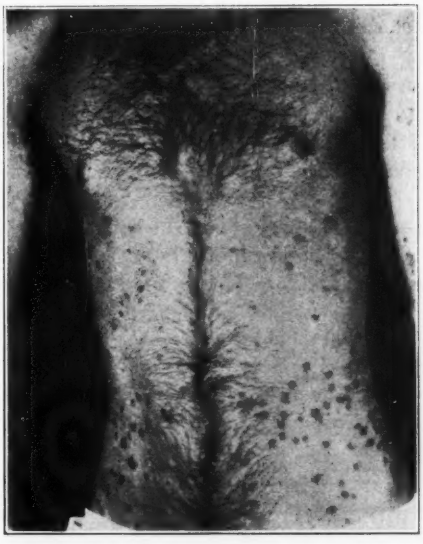

Fig. 4.—Typical flea bites. Note resemblance to papular urticaria. (Courtesy of Samuel Ayres Jr., M.D., Los Angeles.)

furuncles, especially in children, have been observed. Less frequently occur bullous eruptions on the lower extremities and generalized, erythema-multiforme-like, bullous eruptions.

DIFFERENTIAL DIAGNOSIS

It is the experience of those in our office that most cases diagnosed as "papular urticaria" in our district are in reality examples of flea bites. Chipman [4] recognized this fact long ago. While my associates and I do

4. Chipman, E. D.: Urticaria, with Special References to the Cause in the Papular Forms of Children, California State J. Med. **8**:207 (June) 1910.

not deny the existence of true papular urticaria of endogenous origin we are certain that it is rare in comparison with the exogenously flea-produced variety. Children, particularly, or adults who are newcomers to the Coast are sometimes referred to us by pediatricians or general physicians, with the diagnosis of "hives." The story will invariably be that the patient had not been subject to hives prior to coming to the Coast and that the eruption appeared shortly after arrival here. Usually the physician or the patient considers the condition to be an allergy to some of the foods of the West Coast, such as sea foods or fruit. It takes a great deal of persuasion for one to convince these patients that the origin of the eruption is not what they are eating but, rather, what is eating them. Although this fact is known to all the local dermatologists, it is astonishing that it is not known to all physicians and to the laity.

This failure to recognize such a simple, obvious thing as flea bites is not limited to our locality. The Jadassohn [5] school in Germany, Hallam,[6] Tate,[7] and Kinnear [8] in England and Urbach [9] in Vienna reported that papular urticaria in children disappeared when the patients were hospitalized and recurred when they returned home. Reasoning from this information the English observers [10] and Urbach [9] expressed the conclusion that papular urticaria is an allergic manifestation, the exciting cause of which is something in the home environment, food or bedding. These authors made little or no mention of the possibility of fleas or other insects being etiologic factors. One of Jadassohn's pupils, Gerard Simon,[5] described a case of prurigo which cleared while in the hospital and had an exacerbation when the patient returned home. Hallam [6] quoted Jadassohn to the effect that he had noted many years ago that cases of Hebra's prurigo cleared up when the patients were hospitalized. Although the evidence against fleas in these older reports is uncertain, the possibility should be considered. In recent reports the evidence against fleas is much stronger. Hallam [6] reported a series of patients with papular urticaria whose condition cleared on hospitalization only to relapse on their return to their homes. He concluded that papular

5. Simon, G.: Beiträge zur Kenntniss der "Neurodermitiden," Bern, Stämpfli & Co., 1898.

6. Hallam, R.: Further Observations on the Aetiology of Papular Urticaria (Lichen Urticatus), Brit. J. Dermat. & Syph. **44**:117 (March) 1932.

7. Tate, B. C.: Papular Urticaria (Lichen Urticatus), Arch. Dis. Childhood **10**:27 (Feb.) 1935.

8. Kinnear, J.: Urticaria Papulosa, Brit. J. Dermat. & Syph. **45**:65 (Feb.) 1933.

9. Urbach, E.: Skin Diseases and Nutrition, Including the Dermatoses of Children, Vienna, Wilhelm Maudrich, 1932, p. 175.

10. Hallam.[6] Tate.[7] Kinnear.[8]

urticaria is an allergic manifestation, the exciting cause of which is associated with home sleeping arrangements and can be controlled by detaining the child in the hospital for the night only. Kinnear [8] described 14 cases of papular urticaria in children which began on their arrival in England from America and said that other cases occurred in their families. He had also observed that same occurrence in newly arrived medical students. He agreed with Hallam [6] that this condition had something to do with home sleeping conditions. He had no idea of the cause and made no mention of the possibility of flea or other insect bites. Tate [7] said, "The exciting agent is something connected with the patient's home environment."

My associate, Dr. H. J. Templeton,[11] personally observed many cases of "papular urticaria" in Vienna. He reported that patients with so-called papular urticaria suffered from typical flea bites and that their lesions would have been so diagnosed by any of the West Coast dermatologists. He observed this "papular urticaria" developed in members of his own family and also in persons in the families of other American physicians and that the lesions were typical flea bites. That the diagnosis was not shared by all Viennese dermatologists is evidenced by Dietrich's [12] discussion in which he stated that the cases were really examples of insect bites. It is my belief that this confusion occurs in many cases in the literature.

Urbach [13] devoted eight pages to the idea that most cases of papular urticaria are of allergic, endogenous or gastrointestinal origin and only two lines to cases in which insects were the cause. His photographs 347 and 353 [13] are highly suggestive of bites. In another text [9] he showed a photograph of papular urticaria which looks to me like insect bites. Macleod's text [14] has a photograph which is almost certainly one of flea bites, with the puncta, papules, grouping and location. A photograph in Andrews' text [15] needs to be differentiated from one of flea bites. At a clinical meeting of the American Dermatological Association [16] a 3 year old child was presented from Canada as having an example of papular urticaria. In the discussion Ayres and Weiss diagnosed the

11. Templeton, H. J.: Personal communication to the author.

12. Dietrich, A.: Lichen urticatus exogenes, Wien. klin. Wchnschr. **50**:1730 (Dec. 17) 1937.

13. Urbach, E., and Gottlieb, P. M.: Allergy, New York, Grune & Stratton, Inc., 1943, pp. 884-891.

14. Macleod, J. M. H.: Diseases of the Skin, New York, Paul B. Hoeber, 1924, p. 698.

15. Andrews, G. C.: Diseases of the Skin, Philadelphia, W. B. Saunders Company, 1930, p. 348.

16. King-Smith, D.; Trow, E. J., and Dixon, H. A.: Urticaria (Papular), Arch. Dermat. & Syph. **25**:172 (Jan.) 1932.

case as one of insect bites. In the *British Journal of Dermatology and Syphilis* [17] a reviewer wrote of "papular urticaria (a condition furtively believed by not a few since Jonathan Hutchinson to be produced by insect bites)." Jonathan Hutchinson [18] expressed the opinion that fleas had something to do with the lesions: "It may be that flea bites acting on a skin sensitized by faulty alimentary metabolism is the determining cause." Concerning the first step in treatment MacKenna [19] said, "Inquire judiciously into the possibility of infestation with fleas."

From all this evidence, I conclude that elsewhere in the world there is the same problem of differentiating papular urticaria from flea bites that exists on the West Coast.

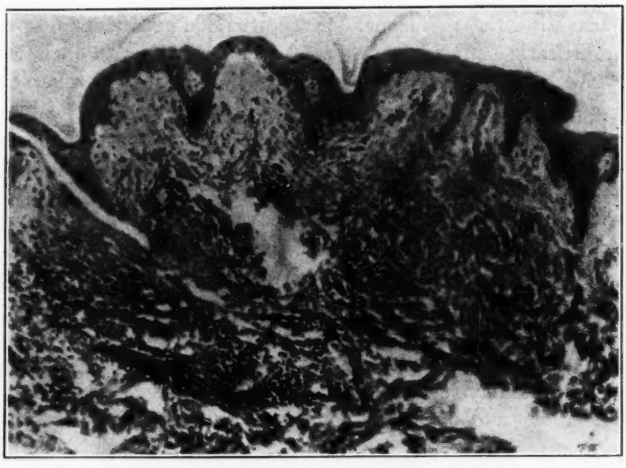

Fig. 5.—Histologic aspects of the flea bite. Note the intensity of the reaction.

PATHOLOGIC ASPECTS

A flea bite papule was excised from each of 5 patients for microscopic examination. Each of these lesions was of the papular urticarial variety most commonly seen as the result of flea bites. None was of the rarer bullous variety.

In none of the excised specimens was there any significant change in the epidermis. Most of the abnormal observations were in the subpapillary and deeper portions of the corium (fig. 5). These consisted

17. Hartman, M.: The Use of Hormones in Dermatology, Brit. J. Dermat. **60**:30 (Jan.) 1948.

18. Hutchinson, J., cited by MacKenna, R. W.: Diseases of the Skin, ed. 3, Baltimore, William Wood & Company, 1933, p. 278.

19. MacKenna.[18]

in a lymphocytic infiltration around the blood vessels, which were dilated and often filled with the lymphocytes. The same lymphocytic infiltration occurred around the sweat and sebaceous glands and, in 1 case, around the muscle fibers. In three of the specimens there was a very pronounced lymphocytic infiltrate around the sweat glands deep in the corium and even as deep as into the fat layer (fig. 6). I was impressed by the intensity of this deep lymphocytic reaction.

DATA OBTAINED FROM OUR LETTERS OF INQUIRY

In the fall of 1947 my associates and I mailed letters to all certified dermatologists in the United States, Canada, Hawaii and Cuba, asking about the flea problem in their communities.

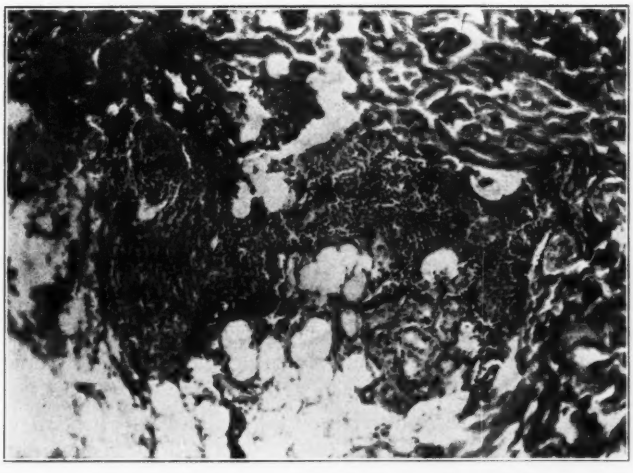

Fig. 6.—Histologic changes seen in the flea bite. Note the intensity and the depth of involvement in the fat layer.

The data in the letters received showed that fleas are a major dermatologic problem only in California. Of the several hundred answers from all other parts of the country only a few persons gave as their opinion that fleas were a major medical problem in their community. A slightly larger, but still small, percentage of letters stated that fleas were a minor problem. In two states, Nevada and Colorado, some observers denied that cat and dog fleas even existed there.

In California, however, in all letters from the San Francisco Bay area there was agreement that a real problem exists. In eight letters from Los Angeles four dermatologists stated that the problem exists and four denied it. All six letters from the Los Angeles suburbs contained statements that a moderate problem exists. From the San Diego

area 80 per cent of the letters had claims that fleas are a real problem. Writers from Pasadena, Santa Barbara, Fresno and San Jose disclaimed that there is a flea problem in their localities.

The writers described about the same varieties of eruptions as I have described. A few specifically mentioned papular urticaria in children.

Flea Antigen.—The great majority of the men stated they had had no experience with the use of flea antigen. Most of those who had used it were from California, and their opinions varied from those of its being worthless to others that it was satisfactory in 70 to 80 per cent of cases treated. Several men observed that the results were highly successful in some cases and that treatment completely failed in others.

Results Reported from the Use of Locally Applied Repellents.—The best results reported from the use of locally applied repellents and insecticides were obtained by those persons who used 0.5 to 10 per cent DDT (2,2-bis[*p*-chlorophenyl] 1,1,1-trichloroethane). Many military observers felt that Army 612 (2-ethyl-hexanediol-1,3) is, on the whole satisfactory. Others reported varying results from the use of pyrethrum, rotenone and sulfur powder.

Reports on the Use of Thiamine Hydrochloride.—Many of the men had used thiamine hydrochloride given by mouth as a repellent with varied results. Eder [20] reported good results in both prevention and treatment. Two reports from San Francisco had observations that thiamine hydrochloride administered internally was a 100 per cent effective repellent of fleas in human beings, cats and dogs. According to others, it was of questionable value or completely worthless.

Selectivity of Fleas.—Several of the men called attention to the selectivity of the action of fleas. That is, very often in a family fleas will bite one member and not another, children being the most frequent victims. Those in our office agree that this observation is an accurate one.

Some of the men were attacked by human fleas but not by fleas from their own dogs. Several observers were attacked by fleas in one geographic location but not in another. For instance, fleas in their place of residence in San Francisco did not annoy them, but when they visited Los Angeles or some foreign city, such as Mexico City or Vienna, they would be severely bitten, the reaction in these cases being described as a severe bullous eruption.

Some of the men thought that such things as body odor and sex hormones should be considered as accounting for the age and sex selectivity.

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20. Eder, H. L.: Flea Bites: Prevention and Treatment with Thiamin Chloride, Arch. Pediat. **62**:300 (July) 1945.

COMMENTS AND STUDIES ON IMMUNOLOGY

The entomologists say that the old idea of host specificity is erroneous. Persons who think they are not bitten by fleas from their own dogs actually may have been bitten until they have established an immunity. Also, some persons not bothered by fleas at home may be sensitive to a slight variation in antigen from fleas in a strange city.

It appears that as a person continues to be bitten he undergoes a natural desensitization to the antigen deposited by the flea. This takes place gradually over a period of about a year. There are people, however, in whom this desensitization occurs more slowly, and in some cases it never occurs. Patients have been known to leave the Bay region permanently because the fleas have made their lives intolerable.

The biologic explanation for the phenomenon of natural desensitization against insects is illustrated by an experiment done by Kenneth Mellanby.[21] To study the varying reactions to insect bites in man he fed the yellow fever mosquito on Englishmen who had never been away from the British Isles. The first bites produced an erythema about 1 to 2 mm. in diameter and in twenty-four to forty-eight hours a delayed reaction consisting of an intensely pruritic urticaria-like papule 1 cm. in diameter surrounded by a 2 to 3 cm. zone of redness. After the subjects had been bitten for a month the immediate reaction was an itching wheal which was followed by the delayed reaction in twenty-four to forty-eight hours. The later reaction recurred each three to four days. As the experiment continued the delayed reaction gradually disappeared and there was less itching, but the immediate reaction never failed to appear. Dr. Mellanby stated, however, that many Englishmen bitten by thousands of indigenous mosquitoes finally lost the immediate reaction and had complete desensitization. He expressed the opinion that the two reactions were quite distinct and were caused by different antigens in the saliva of the mosquito.

My associates and I believe our clinical observations tend to show that a similar process of desensitization occurs naturally on prolonged exposure to flea bites.

In 1939 Cherney, Wheeler and Reed[22] tested two groups of persons to an antigen made from dog, cat and human fleas indigenous to San Francisco. About 85 per cent of the immune group gave no reactions. A number of susceptible persons were given weekly injections for six weeks with the flea antigen in an attempt at desensitization. The results of treatment were fair.

21. Mellanby, K.: Man's Reaction to Mosquito Bites, Nature, London **158**:554 (Oct. 19) 1946.

22. Cherney, L. S.; Wheeler, C. M., and Reed, A. C.: Flea-Antigen in Prevention of Flea Bites, Am. J. Trop. Med. **19**:327 (July) 1939.

In 1941 McIvor and Cherney [23] treated persons sensitive to flea bites by injecting into them an antigen extracted from whole San Francisco fleas. They reported encouraging results.

On the basis of data shown in McIvor and Cherney's article the Lilly Research Laboratory prepared an antigen according to the methods developed by the authors but obtained dog, cat and human fleas from Bolivia and Mexico City. This extract has been used in our office since it was first manufactured in the treatment of children who were most persistently susceptible to flea bites. The results obtained were not controlled and were difficult to evaluate properly because we could not determine whether the "cure" was the result of the injections or was an expression of a natural immunity developed from the patient's being constantly bitten by fleas.

Hatoff,[24] using Lilly's commercial antigen and a technic similar to that used by McIvor and Cherney,[23] reported successful desensitization in about four fifths (80 per cent) of children treated.

We feel that studies should be made to determine whether or not there is a difference in the antigen injected by fleas of different kinds and from different localities and, if so, of what clinical importance it is.

EXPERIMENTS WITH FLEA ANTIGEN

With the cooperation of Drs. Bernard H. Winston of Kansas City, Mo., and Knox Freytag at the Presidio in San Francisco, together with the residents at Highland–Alameda County Hospital in Oakland, 45 children and 104 adults were tested with the Lilly flea antigen, the tuberculin technic being used. Results are so lacking in uniformity that no conclusions can be drawn. Therefore, we wish to reserve our findings until they have been rechecked.

STUDY OF REACTIONS TO FLEA BITES

It is difficult for one to obtain fleas because of the present day universal use and efficiency of DDT as an exterminator. The fleas we have used have been cat fleas, obtained from dogs.[25] These fleas were fed on the forearms of volunteers. The hungry fleas fed without hesitation. When the fleas were satisfied they became active. We usually used six to twelve fleas on each subject. At times we could get only three or four. The fleas will feed three or four times a day and can be kept alive in the test tube with such feedings on the average of only ten to fourteen days.

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23. McIvor, B. C., and Cherney, L. S.: Studies in Insect Bite Desensitization, Am. J. Trop. Med. **21**:493 (May) 1941.

24. Hatoff, A.: Desensitization to Insect Bites, J. A. M. A. **130**:850 (March 30) 1946.

25. The fleas used in our experiments were furnished through the courtesy of Morris A. Stewart, Ph.D.

EXPERIMENTS

With Normal Conditions.—EXPERIMENT 1. The fleas were fed on 12 members of our office staff and their families. All except 1 were old residents of the Bay region. The exception had been reared on a ranch and knew that she had been exposed to fleas all her life. Although the fleas fed with equal avidity on all 12 subjects, only 2 persons showed a positive reaction. Both of these had been flea-sensitive all their lives. The reactions were both immediate and delayed. This experiment demonstrates the immunity of 10 out of 12 old time Californians to flea bites.

EXPERIMENT 2.—Fleas were fed on 8 volunteer students at the University of California in Berkeley, none of whom gave a history of previous sensitivity to fleas. Of the 7 that returned for observation only 1 had been a resident of the San Francisco Bay region for longer than two months. The exception had lived here for six months. Seven of the 8 volunteers showed a positive delayed reaction at the end of forty-eight hours. This experiment indicates the sensitivity of recent residents in the Bay region to flea bites.

EXPERIMENT 3.—Fleas were fed on 4 bottle-fed newborn babies at the Highland-Alameda County Hospital. None of the babies had been out of the hospital. At the end of forty-eight hours there had been no reaction. This experiment indicates that persons on first being bitten by fleas are not sensitive to their bites.

With Local Application of Repellents.—Oil of Citronella: Two of our nurses applied oil of citronella to their forearms. The fleas were repelled and did not feed. It was interesting to see them dance all over the feeding ground.

Pyrethrum: Two subjects had powder containing 0.7 per cent pyrethrum dusted on their forearms. In 1 case the fleas fed. In the other the fleas were repelled after five minutes of feeding. In both instances fleas were listless for twenty-four hours and would not feed but recovered. In 2 other subjects other fleas were exposed to a heavier powder sprinkling. Although we did not see them feed, they all died in twenty-four hours.

Army 612 (2-ethyl-hexanediol-1, 3): Three subjects were tested. The fleas were repelled, and most died within twenty-four hours.

DDT Powder (7.5 per cent DDT): Two subjects were studied. The fleas would not feed after five minutes and died in four hours. We did not pursue this experiment because we could not afford to kill more of the fleas.

Camphor: Spirits of camphor was applied to the forearms of 6 subjects, and fleas were placed thereon. The fleas would not feed. One volunteer forgot to cleanse the camphor from his forearm properly. Four hours later two tubes of fleas were fed on the same site. The fleas were dead in fifteen minutes.

Petrolatum: In four instances fleas would not feed through petrolatum rubbed on the forearm.

With Internally Administered Repellents.—Sulfur: Four patients were fed calcium sulfide, ¼ grain (15 mg.) twice a day for seven days. Fleas fed normally and were not made ill. We have been advised that farmers in certain parts of the country, believing that sulfur will protect them from fleas, take it in the spring for that purpose.

Quinine: Eight tubes of fleas were fed on 4 subjects given 5 grains (0.30 Gm.) of quinine sulfate three times a day for three days. At the end of thirty-six hours 90 per cent of the fleas were dead.

Thiamine Hydrochloride: One subject was given 100 mg. of thiamine hydrochloride, intramuscularly. One half hour later the fleas fed. Six hours, ten hours

and twenty-two hours later, fleas were repelled and would not feed on either the subject or 4 other subjects who had not taken thiamine hydrochloride. Two days later the fleas fed on the original subject.

Four subjects were given 100 mg. of the drug intramuscularly at 11 a. m., 4:30 p. m. and 10:30 a. m. the next day. At 1 p. m. fleas were planted on all subjects and fed for five minutes, and then they quit feeding. At 8 a. m., 9 a. m. and 3 p. m. the next day the fleas would not feed. At 8:30 a. m. the third day most of the fleas were dead. The fleas that remained alive fed.

Seven test tubes of fleas were fed on 3 subjects given 100 mg. of thiamine hydrochloride by mouth, three times a day. At the end of six hours the fleas fed reluctantly. At subsequent plantings they fed for only five minutes and were inactive. At the end of three days most of them were dead.

This experiment indicates that at first fleas are repelled but later feed and die when they are planted on patients given thiamine hydrochloride.

Fleas were fed on 4 persons with a history of sensitivity to fleas. There developed a typical flea bite reaction. After the subjects had taken three doses of 100 mg. of thiamine hydrochloride, the fleas were again fed on them. The subsequent effect was either a less severe reaction or none at all.

The question arises as to whether this lowered reactivity was due to the desensitizing or to the repellent action of thiamine hydrochloride.

CONTROL OF FLEAS

The most practical method of controlling the flea problem is by means of the newer insecticides, such as DDT. If this substance is sprayed onto carpets, floors, overstuffed chairs and basements, the fleas will be quickly destroyed. It is not necessary for one to apply it to dogs and cats to rid them of fleas. Instead it should be sprayed into their kennels or nests or onto their favorite sleeping places. One such spraying every two months will keep animals free from fleas.

CONCLUSIONS

Flea bites constitute an annoying problem in coastal California, particularly to newcomers in the San Francisco Bay area.

My associates' and my experiment of feeding fleas on newborn infants confirms our clinical observations that persons first have to be bitten by fleas in order to become sensitive. Our experiments of feeding fleas on old time Californians and on newcomers confirm our clinical findings that most people become immune after having been bitten over long periods.

We are certain that most cases of "papular urticaria" in California are, in reality, due to flea bites and suspect that this is probably true elsewhere.

Further studies and better antigens are needed for the investigation of the value of artificial desensitization.

Oil of citronella, pyrethrum, Army 612, DDT, petrolatum and camphor applied locally are of value as repellents.

The continued taking of quinine by mouth or of thiamine hydrochloride by mouth or injection is of clinical and experimental value in the repelling of fleas.

Modern insecticides, such as DDT, applied to the known breeding places of fleas offer the most practical solution of the problem.

3115 Webster Street (9).

ABSTRACT OF DISCUSSION

DR. LESLIE M. SMITH, El Paso, Texas: I spent the summer of 1925 in the clinic of Dr. Harry Alderson at Stanford, and I have had both clinical and personal experience with the hardy California fleas. I have seen reactions to their bite so severe and extensive as to suggest erythema multiforme or urticaria. That allergy to the toxins injected by fleas and other insects occurs in some persons there can be little doubt. This is evidenced by the extreme local reactions which occur in some victims and, I believe, by the appearance of some urticarial lesions which do not occur at the sites of bites, or at least do not contain the puncta of typical bites.

In the El Paso area, on account of the very low humidity, fleas are no problem. There are mites, bedbugs, mosquitoes and some other insects, and manifestations of allergy are occasionally observed following the bites of some of these. In addition to typical bites one occasionally sees scattered urticaria papules, which do not appear to be the result of bites and presumably are an allergic response to absorption of toxin from the bitten areas.

I am not prepared to state that in patients encountered in my practice insect bites are the principal cause of lichen-urticatus-like eruptions, but I think it is quite probable that I have not been sufficiently alert to the possibility of this factor in such cases. It is a common observation that most cases of lichen urticatus and prurigo occur among the type of patients in whom malnutrition is common. These same patients usually live under conditions which favor the presence of insects and the possibility of insect bites. I, for one, shall be more conscious hereafter of the possible role of insects in the lichenoid and urticarial papular eruptions.

I have had no experience with treatment by desensitization to insect toxins, but the fact that a person may gain tolerance after being repeatedly bitten would seem to indicate that tolerance might be produced artificially by hypodermic injections of the proper antigen. I believe this line of reasoning is worthy of further work.

In areas where fleas constitute such a problem as they do in California, it is probably wise to give orally or parenterally administered repellents, such as quinine or thiamine, to susceptible persons, but where fleas and other insects are not so numerous I believe the intelligent use of repellent sprays about their breeding places and about the patient's environment will be sufficient for their control.

I should like to know what success Dr. Lunsford has had with use of the antihistaminic drugs in the control of reactions to insect bites.

DR. PAUL E. BECHET, Elizabeth, N. J.: Dr. Lunsford's paper is of interest, in view of the fact that plague-infected fleas (Xenopsylla cheopis) caused the unprecedented death of over 60,000,000 persons from the year 1348 to the end of the Seventeenth Century. Another killer of men, typhus, can be carried by fleas as well as by lice, which observation calls attention to the fact that Hieronymus Fracastorius, the author of "Syphilis sive Morbus Gallicus" published "Contagion,

Contagious Diseases and Their Treatment" in 1546, and in his discussion of the etiology of typhus stated that it is caused by a "germinis" transmitted to man by the bite of an insect and not by direct contact or fomites. This brilliant observation of Fracastor's four centuries ago was unfortunately completely ignored; had it been acted on and further investigated, it would have saved millions of lives.

I hope I may be pardoned for bringing up the lethal importance of the flea, rather than its dermatologic significance, and also that Dr. Lunsford may not suffer for his temerity by mentioning fleas and California in the same breath.

Dr. N. P. Anderson, Los Angeles: When I first came to California I mistakenly diagnosed flea bites as ordinary urticaria. Occasionally the diagnosis of flea bites is made on the findings of bullous lesions the size of a walnut. It is hard to conceive that such bullae can be the results of flea bites. I think that Dr. Lunsford, having access to so many fleas, could disprove the idea that a great many of these lesions are allergic. It seems to me that this should be proved or disproved. Take these fleas; isolate the person, and let one flea bite him to see whether lesions remote from the bite do occur. I don't believe that these so-called remote lesions are allergic. I have seen five hundred lesions on one member of my family. At certain times of the year there is an epidemic of flea bites. Such an epidemic is usually associated with hot spells. Endemic cases are seen throughout the year. I should also like to mention the transmission of disease by biting insects. The bubonic plague first appeared in this country in the Chinese quarter of San Francisco early in the 1900's. Oriental fleas carrying the transmittal agent of bubonic plague have been found as far east as Colorado. This reservoir of plague extends over the nation and sometime in the future may burst forth into an epidemic.

Dr. Norman Epstein, San Francisco: I had the pleasure of reading Dr. Lunsford's paper at home, and I think he should be complimented on the well planned investigation which he has made. Examination of the literature shows a lack of papers on this subject. Those of us who practice dermatology in the San Francisco Bay area will concur in the conclusions which Dr. Lunsford has drawn. There may be some difference of opinion about certain points. Dr. Anderson has emphasized the role of the flea in transmitting diseases. We are all familiar with that. Some of us do not realize that bubonic plague, which is transmitted by the flea, still exists in this country. There have been 506 cases of bubonic plague in America since 1900. There have been 300 reported from San Francisco, in 268 of which the patient died. The last case was reported in 1942, but we cannot be assured that new cases will not develop when least expected. Tularemia is spreading from California throughout the country and into Canada by way of fleas transmitted from one rodent to another. I agree that a large majority of the cases of papular urticaria in San Francisco are very likely due to flea bites. At times the eruption is so severe that some people have to move from San Francisco. One of the most remarkable things about the flea is that it will not bite everyone. This selectivity on the part of the parasite can hardly be explained on desensitization alone. The work of Shannon in regard to thiamine hydrochloride in the repelling of mosquitoes may have a practical bearing in making certain parts of the world more habitable. This work was done in 1933. Eder noted that fleas were repelled from children given thiamine hydrochloride. Shannon expressed the belief that this action was due to a change in body odor of the person taking the thiamine hydrochloride. Perhaps that is the explanation. Usually one sees fleas on logs and flea bites on patients who work in warehouses on wooden floors. The lesions are occasionally bullous.

In our hands, the use of flea antigen has not been convincing in its effects. My associates and I have been unable to say that flea antigen has accomplished very much clinically. In our hands, the antihistaminic drugs have given some relief.

DR. SAMUEL AYRES JR., Los Angeles: With reference to humidity, it seems that in our part of the state the flea season is in the hot, dry months, beginning with July, August and September, when the humidity is low. The definite instances of flea bites are more pronounced at this time. The question of regional immunity is another interesting thing. People reaching the Bay area become bitten, and I have also encountered long time residents of San Francisco who have been bitten by our variety of flea. One can be immune to California fleas but not to Mexican fleas. People may be bitten by cat and dog fleas in any part of the country. I know of one person who has been susceptible to dog fleas and is unable to keep dogs. Several years ago, in the literature, I ran across an account of an experiment in which a flea was put in the center of a round table and invariably hopped to one person regardless of his position at the table. I have had some degree of success with the use of histamine azoprotein (hapamine®) desensitization for highly sensitive persons. This drug did not stop the fleas from biting, but the reaction was much less severe. An editorial in the *California Medicine* about eight months ago (Hartman, M. M.: *Calif. Med.* **60**:242, 1947) mentioned the valuable effect of histamine azoprotein in treatment of insect bites. Thiamine hydrochloride in a dose of 100 mg. three times a day is of definite value in conferring temporary immunity.

DR. SAMUEL PECK, New York: We have had relatively little experience with flea bites in New York city, but, if I may judge on analogy with the investigations which my associates and I carried out during the War ("Cutaneous Reaction Due to the Body Louse," *J. A. M. A.* **123**:821 [Nov. 27] 1943), it is probable that the flea bite, like the louse bite, is an allergic reaction. Pruritus became evident only after allergy was established.

Interestingly enough, we could show that the feces as well as the bite of the louse played a role in the cutaneous eruptions which were produced.

DR. C. F. LEHMANN, San Antonio, Texas: Since the question of allergy is brought up, I rise to testify that I have remained sensitized to the toxin of the flea bites since I was a small boy and was heavily infested with fleas while seeking shelter from a heavy rainstorm one night. Ever since then, a flea bite will cause an urticarial reaction not only locally but also generally in me.

The diagnosis of such bites sometimes presents quite a problem. A child may be brought in with a line of papules with central puncta. The flea has the propensity of crawling along a line and biting, and the line of papules is strong corroborative evidence of flea bites, despite the fact that the proud mother might not want to admit such things as fleas having bitten her child. These lines of papules are sufficient evidence to substantiate the suspicion of the etiologic factor.

DR. H. S. ALDEN, Atlanta, Ga.: I don't believe that flea bites occur only in California. We have them in Georgia. In Georgia the "chigger flea" produces most of our troubles. I should like to mention the fact that the Negro (the dark Negro, not the mulatto) is more or less immune to the bites of the chigger. White men could not be worked in the swamps, but the Negroes could. Once I tried to get some students to collect chiggers by laying a sheet or some white material on the ground. The chiggers will come to that sheet in large quantities and can be collected. I have therefore warned patients never to go into the woods with white shoes or white clothes.

DR. C. J. LUNSFORD, Oakland, Calif.: I thank all the discussers for their expressed interest in my paper.

In answer to Dr. Leslie Smith's question: I have not had sufficient experience with the use of the antihistaminic drugs in the control of reactions to flea bites to permit me to discuss the subject.

In answer to Dr. Bechet's question as to the part blood-sucking insects play in producing the "cutaneous ills of mankind," my observations have in the main been limited to the bites of fleas. In those cases in which the allergic reactions are expressed as generalized urticarial or erythema-multiforme-like eruptions, I think there is no doubt but that a rise in temperature would be present.

Dr. Anderson's idea of planting fleas on persons suspected of being allergic to flea bites and thus demonstrating whether or not such lesions as bullous ones develop far from the sites of the bites is an interesting one. My associates and I hope to be able to follow through on it.

I was interested in Dr. Peck's experiment, proving that in the case of the louse the antigen producing the cutaneous reaction was obtained from the feces as well as from the salivary glands of the louse. The group from the Hooper Institute, who did the original work in preparation of the antigen used by Eli Lilly, ground up the whole fleas, including the contents of the intestinal tract, in the production of their antigen. In the case of fleas, however, the clinical observation of inflammatory reaction developing about the central punctum would make it appear likely that the flea antigen is in their salivary glands.

We were interested to learn that Drs. Epstein, Ayres and Lehmann agreed with us that fleas can produce an allergic response.

Clinical Notes

PAIN IN A BREAST DUE TO NEUROSYPHILIS

CARL F. BAUMEISTER, M.D.
RIVERSIDE, ILL.
VICTOR P. SLEPIKAS, M.D.
BERWYN, ILL.
AND
DUANE D. DARLING, M.D.
CHICAGO

ANY NEW manifestation of syphilis is a matter of clinical interest. The literature fails to reveal an authenticated instance of neurosyphilis as a cause of pain in a breast.

REPORT OF A CASE

Mrs. K. D., aged 55, had pain in the right breast, increasing in amount for five years. This pain would tend to come in waves of sudden onset, severe intensity and varying duration of one to thirty minutes and would terminate with a feeling of soreness. The number of attacks in twenty-four hours would vary, but they were tending to occur almost continuously in the month prior to her hospitalization, which occurred in February 1947. When the patient had an attack and was not taking opiates she could obtain relief only by squeezing her breast violently. Since this was not very effectual, codeine or other opiates were given four or five times daily, with partial relief.

Since the pain was equally severe and bizarre, the patient had seen numerous physicians, and very complete work-ups, including many serologic tests, had been made. Owing to the fact that she could not speak English well and tended to call her bouts "gas pains," her appendix had been removed in 1943. In April 1946, because the gallbladder was poorly visualized, it was removed; the pathologist reported mild fibrosis. Neither of these procedures was of the slightest benefit as far as the main symptom was concerned. The situation had reached the point that the patient wanted her breast removed.

For the last two years she had been having increasing difficulty in vision, tending to have diplopia at times in the last year. She had had three normal children, who were all living and well.

There were only a few points of pertinent interest in the examination: 1. The patient had paresis of the right sixth cranial nerve. 2. The reaction to light was sluggish in both eyes but especially in the right. 3. The breasts revealed no abnormality. 4. Other neurologic findings were not significant.

As the patient had had complete gastrointestinal and cardiopulmonary examinations, with roentgenograms and electrocardiograms proving noninformative, it was decided to see if we were not dealing with one of Head's zones of referred pain. Accordingly, a lumbar puncture was done for the first time on this patient on Feb. 8, 1947; there was no increase in pressure. One specimen of spinal fluid examined in the hospital laboratory gave a negative result in the Kahn test; the globulin content was 2 plus, and the Lange colloidal gold curve was 5543221000. A portion of the same specimen sent to the state laboratory gave a positive reaction to the Kahn test; there was no report on globulin, and the colloidal gold curve was 1123322110. As usual, all the serologic tests showed negative results.

A tentative diagnosis of neurosyphilis was made, and, starting on February 15, an injection of iodobismitol® (a solution of bismuth sodium iodide and sodium iodide in propylene glycol containing saligenin and acetic acid) was given twice weekly on eight occasions. Within three weeks the patient noted a definite diminution of the pain in the breast. A total of four injections, each of 2 Gm., of tryparsamide U. S. P., was given weekly, starting March 19. This therapy was discontinued because, though there was almost complete disappearance of the pain, the vision became much poorer. The patient had another course of injections of iodobismitol,® followed by four weekly injections of 45 mg. of dichlorophenarsine hydrochloride U. S. P. On June 18 spinal fluid obtained by lumbar puncture revealed 3 plus globulin and gave a negative result in the Kahn test and a colloidal gold curve of 0011211000.

The patient refused to see an ophthalmologist. In June, when an attempt was made to transfer her to a syphilologist nearer her home, treatment was discontinued.

CONCLUSION

A case of pain in the breast due to neurosyphilis is presented, which is believed to be the first of its kind recorded in the literature.

GRANULOMA CUTIS CALCINOSUM FOLLOWING INJECTION OF CALCIUM LEVULINATE

CHAIM BERLIN, M.D.
TEL-AVIV, ISRAEL

A peculiar local untoward incident following an intravenous injection of calcium levulinate came to my observation recently. At the site of injection a nodular eruption, which took the appearance of granuloma annulare, developed.

REPORT OF A CASE

M. D., a boy aged 17, was admitted to the hospital in July 1945 because of peritoneal tuberculosis accompanied with ascites. He was given roentgen therapy and intravenous injections of calcium levulinate, with much improvement in his condition. Once during such an injection he experienced a painful sensation and slight local redness. The physician, fearing a subcutaneous infiltration, stopped the injection immediately. But no infiltration occurred, and the redness and tenderness subsided within several hours. The next day, a papular eruption appeared at the site of injection. When the patient was seen by me, six days after the incident, the left cubital area presented a plaque measuring 1.5 by 1 cm., consisting of pinhead-sized to match head–sized, slightly raised, firm, closely aggregated and partly coalesced nodules with smooth surfaces. The color was pinkish to yellowish and became lighter on diascopic pressure. The lesions were neither pruritic nor painful. Most of the nodules were arranged in a horseshoe fashion, and the plaque closely resembled the picture of granuloma annulare (fig. 1). No change was observed during the next two weeks. The plaque was then totally excised, and the microscopic examination made by Dr. Karplus confirmed the tentative diagnosis of deposits of calcium. A report on the examination follows:

"Corresponding to the nodules distinctly seen on gross observation, there were foreign body granulomas in the form of tubercles with many giant cells of middle size in the deeper layers of the cutis. Most of the cells were fibroblasts, and there

From the Department of Dermatology, Hadassah Municipal Hospital.

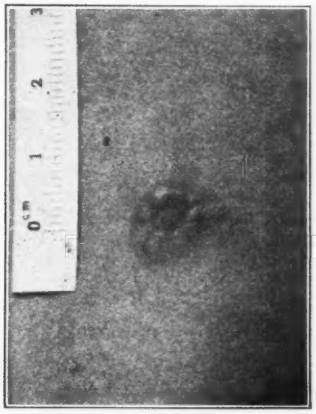

Fig. 1.—Nodular lesions in the cubital area appearing after an intravenous injection of calcium levulinate.

Fig. 2.—Photomicrograph (× 100) showing foreign body tubercles with giant cells and deposits of calcium.

were only a few inflammatory elements. Sections stained with hemalum and eosin revealed in the center broad, calcified, collagenous bundles (fig. 2): The fundamental color of the tubercle was somewhat bluer than was that of the surrounding tissue. A Kóssa stain gave wide impregnation of silver in the fibers throughout the tubercle. The nolular occurrence of the process indicated an embolic process; that is to say, during the injection calcium reached the skin through small arterioles."

COMMENT

This strange form of calcinosis is certainly extremely rare. On the other hand, it is obvious from the history and the histologic observations that the eruption was a consequence of the injection of calcium levulinate. However, nothing definite can be said about the mechanism of the eruption. The passage of a calcium compound outside the vein usually produces severe, nonspecific, painful infiltration. But in this case it must be assumed that even during the puncture particles of calcium succeeded in reaching the skin. There they were dispersed, to cause the formation of reactive nodules.

SUMMARY

A case of calcinosis of the skin following an intravenous injection of calcium levulinate is reported. Around the site of the injection in the cubital fossa an immediate erythema, accompanied with pain, developed. This rapidly subsided and was followed by a group of asymptomatic, small, firm, pinkish nodules, which, with their semicircular arrangement, resembled granuloma annulare. Histologically, calcified collagenous bundles and foreign body giant cells were found.

9 Montefiore.

REACTIONS TO BISMUTH SODIUM TRIGLYCOLLAMATE (BISTRIMATE®)

LOTHAR WIRTH, M.D.
RENSSELAER, N. Y.

Bismuth sodium triglycollamate (bistrimate®), a new oral bismuth compound for oral administration, has been found effective in systemic bismuth therapy.[1] Only minor side reactions, which were limited to the gastrointestinal tract, were observed—reactions which, after dosage had been temporarily reduced, did not interfere with continued administration of bismuth sodium triglycollamate.[2] More recently, however, I saw 2 patients exhibit reactions to bismuth sodium triglycollamate which prevented continuation of treatment with this drug, although bismuth subsalicylate given parenterally was tolerated well. I thought it might be worth while to report these cases.

CASE 1.—A 70 year old man with tertiary syphilis of the central nervous system was started on treatment with bismuth sodium triglycollamate in the recommended dosage of 50 mg. three times daily after meals. On the third day of this regimen he exhibited an edematous lower lip, bluish discoloration of its mucosa

1. Lehman, R. A., and Fassett, D. W.: Experimental and Clinical Studies on Oral Bistrimate (Sodium Bismuth Triglycollamate) for Systemic Bismuth Therapy, Am. J. Syph., Gonor. & Ven. Dis. **31**:640 (Nov.) 1947.

2. Gross, E. R., and Wright, C. S.: Oral Bismuth Therapy in Syphilis and Various Dermatoses, read before the Pennsylvania Medical Society, Philadelphia, Oct. 8, 1946.

and continuous salivation. Food intake was very difficult. Treatment with bismuth sodium triglycollamate was discontinued, and it took about ten days for the symptoms to subside. It was then discovered that the patient had dissolved the tablets of the drug on his tongue, as he could not swallow pills. Treatment was resumed, without ill effect, with parenteral administration of bismuth subsalicylate.

CASE 2.—A 55 year old man with syphilitic valvular heart disease was given bismuth sodium triglycollamate in the usual dosage. He returned two weeks later with a generalized papulopustular eruption, which cleared within three weeks after treatment with the drug was discontinued. Further treatment with bismuth sodium triglycollamate was then tried, at which time the eruption recurred. After one month without therapy, treatment was resumed, without further difficulties, with bismuth subsalicylate administered intramuscularly.

It seems appropriate to instruct patients not to dissolve bismuth sodium triglycollamate on their tongue, as this procedure undoubtedly predisposes to reactions within the oral cavity.

I think the second case is unusual in that a cutaneous eruption occurred in a patient receiving bismuth sodium triglycollamate, while bismuth subsalicylate given parenterally was tolerated well.

82 Broadway.

PENICILLIN-RESISTANT EARLY SYPHILIS

Report of a Case

LOUIS WEXLER, M.D.*
AND
ROBERT N. BUGG, M.D.*
NEW YORK

CAPTAIN HERBERT V. ADAMS
AND
LIEUTENANT COLONEL ALLEN D. SMITH
MEDICAL CORPS, ARMY OF THE UNITED STATES

Since the inauguration of penicillin therapy, resistant early infectious cases have rarely been encountered. To our knowledge only 1 such case has been reported.[1]

It is the purpose of this paper to present a case of penicillin-resistant early syphilis.

REPORT OF A CASE

A 24 year old Negro was admitted to the Dermatology and Syphilology section on Sept. 5, 1946 because of a penile lesion of six weeks' duration. After appearance of the lesion no treatment had been sought for three weeks. At that time the patient had received one intravenous injection of arsphenamine from a private physician, after which treatment the lesion became swollen and painful and did not heal. No further treatment was obtained until his entrance to the hospital. At admission, dark field examination revealed Treponema pallidum

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From the Dermatology and Neurosyphilis Center, Oliver General Hospital, Augusta, Ga.

* Formerly Captain, Medical Corps, Army of the United States.

1. Tyson, W. G.: Early Syphilis Resistant to Treatment with Penicillin: Report of a Case, J. Invest. Dermat. **6**:279 (Oct.) 1945.

and Kahn and Wassermann tests of the blood elicited positive reactions; a diagnosis of primary syphilis was made, and penicillin therapy was started, 100,000 units every three hours for eighty injections. During the period that the patient was receiving therapy there was no improvement in the appearance of the chancre. On the last day of treatment the prepuce became edematous and nonretractable, and concurrently a generalized, papular, erythematous, discrete, symmetric eruption appeared. The papules ranged in size from that of a match head to that of a pea. On the next day the eruption was more pronounced and more generalized. Dark field examination of one of the papular lesions revealed it to contain living T. pallidum, and the titer in the quantitative Kahn test at that time was 20 Kahn units.

The patient was then started on a course of oxophenarsine hydrochloride (mapharsen®) and bismuth subsalicylate injections. Two weeks later, after the patient had received four injections of oxophenarsine hydrochloride of 0.06 Gm. each and two injections of bismuth subsalicylate, 0.2 Gm. each, all cutaneous lesions had disappeared, and the titer in the Kahn test was 3 Kahn units. At that time the patient was discharged with instructions to report to the local treatment center for completion of therapy, and further contact with the patient was lost.

The penicillin used was a crystalline sodium penicillin supplied by the Army and of current stock; the lot number was not recorded at the ward. However, concurrent usage in the cases of other patients with primary and secondary stages of syphilis revealed no further deviation from the expected result.

SUMMARY AND CONCLUSIONS

A case of penicillin-resistant primary syphilis is presented. After having received 8,000,000 units of penicillin, the patient acquired secondary syphilis. After this development the response to heavy metal therapy in this case was very favorable.

34 West Seventy-Fourth Street.

ENDEMIC FAVUS IN TEXAS

BEN R. EPPRIGHT, M.D.
AND
C. H. McCUISTION, M.D.
AUSTIN, TEXAS

A white woman aged about 36, consulted us on Nov. 3, 1947, because of an eruption of the scalp, with loss of hair.

She stated that the disease had begun about one year previously with localized scaliness and mild itching of the scalp. The eruption gradually spread, the scaling became severer, and loss of hair ensued in the areas in which the eruption had been present the longest. Although born in Poland, the patient had come to Texas twenty-five years ago and had not been out of the state since that time. Both the patient and her parents stated that she had had no scalp disease of any kind prior to about a year ago and that no one else in the family had had any disease of the scalp. She knows of no contact in the last few years with anyone from foreign countries.

Examination revealed a woman of about the stated age, somewhat underweight. The entire scalp was involved with scaling and numerous crusts and scutula. The scutula were cup shaped, with the concave side facing upward. Hairs, dull and

lusterless, pierced the crusts and scutula. There was an offensive "mouse" odor to the scalp. There were numerous areas of atrophic scarring with alopecia. Examination by means of the Wood filter revealed a dull gray fluorescence of about 60 per cent of the hairs of the scalp. Of interest was the fact that the involved hairs were fluorescent throughout their length. Several of these hairs were pulled for microscopic examination. The hairs pulled with some difficulty, though with no pain to the patient. Exerting tension on the hair gave one the same impression as pulling a stiff rubber band; i. e., the hairs were distinctly tensile.

Two finger nails were involved slightly, the process beginning at the distal lateral portion of the nail. Microscopic examination of the nail from these areas showed numerous myceliums.

Microscopic examination of hairs treated with a 20 per cent solution of potassium hydroxide revealed large spores, many in long chains and others occurring individually. In crusts clinging to hairs, hyphae were extremely numerous. The spores were seen within the entire length of the hair shaft. Also noted within the shaft were numerous air bubbles.

A tentative diagnosis of favus was made, and material was sent to Dr. E. D. DeLameter of the Mayo Clinic and to Mr. David Mitzkus of the Mycology Laboratory at the Army Area Laboratory, Fort Sam Houston, Texas, for culture. Both laboratories reported growth of Achorion schoenleini.

The patient has refused roentgen epilation, and treatment has consisted of manual epilation, with the use of a General Electric Purple X® bulb for fluorescence and topical application of an ointment of 20 per cent zinc undecylenate with 5 per cent undecylenic acid (zincundecate [desenex®]) and other fungicidal preparations. Most of the improvement noted is apparently due to the correction of the hygienic status of the scalp. At this time the infection is still unchecked.

A questionnaire was sent to several dermatologists throughout the state. Dr. Bedford Shelmire of Dallas stated that he had seen 2 cases of favus, which apparently were endemic, within the past twenty years. Dr. C. F. Lehmann and Dr. J. L. Pipkin of San Antonio, reported that "the only cases of favus which we have seen were outside San Antonio." All others questioned stated that they had never seen a case of favus in Texas.

112 West Seventh St.

CONGENITAL DERMOID INCLUSION CYST

Report of a Case

MAURICE J. COSTELLO, M.D.
NEW YORK
WILLIAM F. LOVEBURY, M.D.
COLUMBUS, OHIO
AND
JOHN F. DALY, M.D.
NEW YORK

Dermoid cysts situated on the bridge of the nose are relatively rare, and while seen occasionally by otolaryngologists they have not been brought to the attention of dermatologists.

This type of cyst belongs to the group of tumors found about the nose which, according to Luongo,[1] result from the trapping of a portion of the dermis by

1. Luongo, R. A.: Dermoid Cyst of the Nasal Dorsum, Arch. Otolaryng. **17**:755-759 (June) 1933.

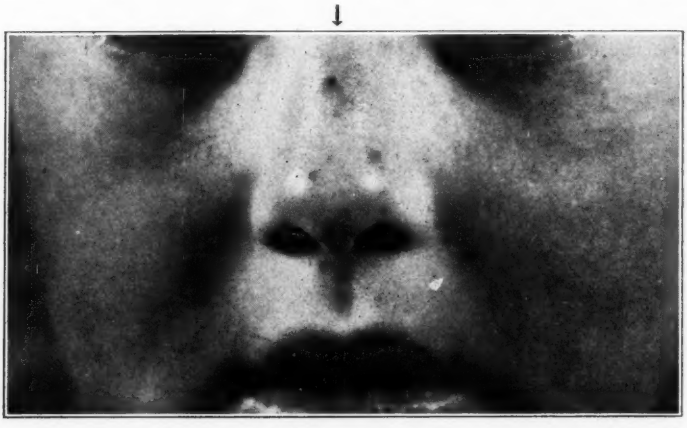

Fig. 1.—Congenital dermoid inclusion cyst of the nose, with protruding short, black hairs.

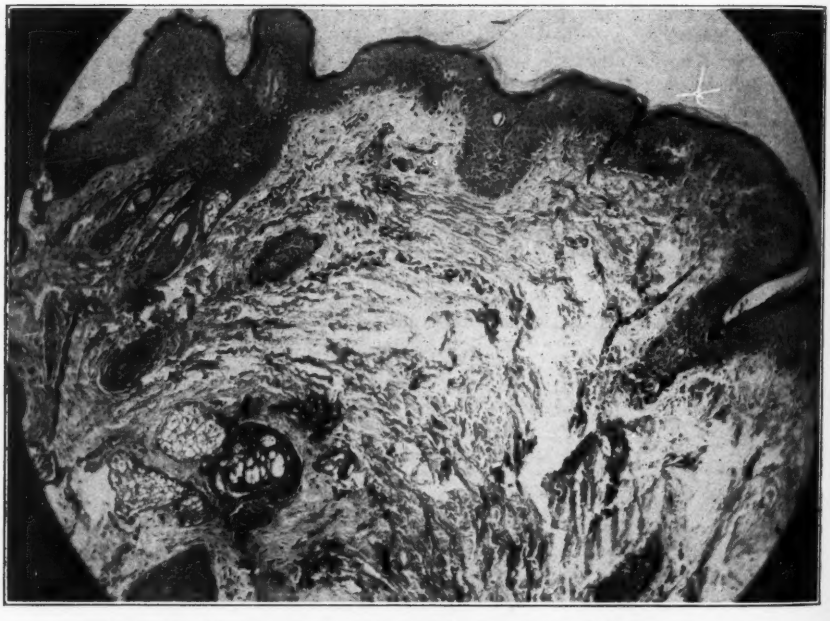

Fig. 2.—Partial view of the epidermis lining the floor of the cyst; low power magnification; hematoxylin and eosin stain.

intrusion of the frontonasal plate between the embryonic nasal dermis and mucosa. The bridge of the nose, exactly in the midline, is a common location.

Most of these growths are discovered before the fifth year of age. Secondary infection is common and is followed by sinus formation, with discharge of sebaceous material and pus. Occasionally, as with the patient considered here, the presence of hairs calls attention to these congenital anomalies.

REPORT OF CASE

A. B., a white girl, was first seen by one of us (M. J. C.) when she was 5 years old. Her mother gave a history of noticing a small lesion on the bridge of the child's nose when she was 6 months old. Since that time the growth had constantly had small black hairs and a thick discharge coming from the opening in the skin. There was no family history of similar lesions or other congenital defects.

Physical examination revealed a well nourished, well developed, white female child with no abnormalities except a small, match head–sized opening on the bridge of the nose at the junction of the cartilaginous and bony portions, exactly in the midline. From this opening thirty to forty black hairs protruded. The hairs were midway between lanugo and terminal adult hairs and could be removed easily with moderate traction. On gentle pressure a thick, sebaceous material could be expressed. There was little pain and no inflammatory reaction around the opening of the sinus.

Treatment, which was carried out by Dr. John F. Daly, consisted in injecting methylene blue into the cyst to facilitate determination of the exact extent of the lesion. An elliptical incision was made around the opening of the sinus, and the growth was completely dissected out. It was found to mushroom out under the skin and was about pea sized, extending down to the junction of the cartilage and bone of the nose, but it did not involve either. Grossly, the lining of the cyst looked like the skin of the scalp or bearded region.

Histopathologic examination of the lining of the cyst revealed a normal-appearing epidermis except for a number of vacuolated cells in the basal layer. The dermis contained no abnormal cellular elements but did show a large number of hair follicles with their associated sebaceous glands. There was no evidence of sweat glands, a finding which has been consistent in previously reported cases.

COMMENT

Congenital dermoid inclusion cysts of the bridge of the nose frequently extend down into the nasal septum and necessitate extensive surgery. Other methods of therapy, such as electrolysis or destruction with high frequency current or cautery, often result in inadequate removal, recurrences and poor cosmetic results. On one occasion, we saw a lesion such as the one described treated as a sebaceous cyst by electrosurgery with a final result of the development of a basal cell epithelioma.

SUMMARY

A case of congenital dermoid inclusion cyst of the nasal dorsum is presented. An excellent cosmetic result, with no recurrences of the growth, followed treatment.

140 East Fifty-Fourth Street (*22*).

LICHEN-SCROFULOSORUM-LIKE LESIONS ASSOCIATED WITH SARCOIDOSIS

Report of a Case

MATTHEW J. BRUNNER, M.D.
AND
MILTON ROBIN, M.D.
CHICAGO

Lichen scrofulosorum is characteristically associated with a high degree of sensitivity to tuberculin, patients in up to 67 per cent of cases showing positive reactions to old tuberculin in a dilution of 1:1,000,000.[1] The finding of apparently typical lesions of lichen scrofulosorum in a patient exhibiting negative reactions to intracutaneous injections of old tuberculin in dilutions as low as 1:10 is therefore unusual. Microscopic examination of cutaneous biopsy specimens in this case revealed a histologic structure characteristic of sarcoid, and on further study laboratory and clinical observations of sarcoidosis involving the eye, salivary glands and lungs were made. The association of lichen scrofulosorum with sarcoidosis has been noted on several occasions in cases cited in the transactions of dermatologic society meetings, but the observation of a sarcoidal histologic pattern in lesions of lichen scrofulosorum is apparently unique. Mitchell and Nomlund,[2] Cannon,[3] Cornbleet,[4] Michelson[5] and Becker[6] have reported on the clinical concomitance of the two conditions. Biopsy of the lichenoid lesions in Cornbleet's case was reported as revealing hematogenous tuberculosis. In Cannon's case, the histologic appearance was that of lichen spinulosus.

REPORT OF CASE

A mulatto woman, aged 26, was seen in the Department of Dermatology, Research and Educational Hospitals, in August 1946, complaining of joint pains, dryness of the mouth, swelling of the glands of the neck and a cutaneous eruption of three months' duration. There had been slight loss of weight, fatigability and low grade fever during this period. Examination on admission to the hospital revealed a slender woman, apparently not acutely ill, with an eruption on the neck, shoulders and back, consisting of grouped, acuminate, pinpoint-sized to millet seed–sized, skin-colored papules, some of which were pierced by short, horny spines. A few of the patches had cleared centrally to produce annular lesions (figure). There were numerous areas, from the size of a dime to that of a quarter,

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From the Department of Dermatology, University of Illinois College of Medicine, Dr. F. E. Senear, Director.

1. Sulzberger, M. B.: Dermatologic Allergy, Springfield, Ill., Charles C Thomas, Publisher, 1942, p. 234.

2. Mitchell, J. H., and Nomlund, R.: Lichenoid Sarcoid and Lichen Scrofulosorum, Arch. Dermat. & Syph. **35**:334 (Feb.) 1937.

3. Cannon, A. B.: Lichen Scrofulosorum and Sarcoid, Arch. Dermat. & Syph. **38**:91 (July) 1938.

4. Cornbleet, T.: Tuberculosis Cutis (Scrofuloderma, Sarcoid and Lichen Scrofulosorum), Arch. Dermat. & Syph. **38**:272 (Aug.) 1938.

5. Michelson, H. E.: Sarcoidosis, Arch. Dermat. & Syph. **46**:774 (Nov.) 1942.

6. Becker, S. W.: Sarcoid (Miliary), Arch. Dermat. & Syph. **48**:223 (Aug.) 1943.

of subcutaneous hemorrhage on both legs and thighs. The oral mucous membranes were dry, and the submaxillary salivary glands were visibly enlarged, firm and of a rubbery consistency. The parotid glands were affected similarly, but to a less degree. Except for a slight hepatic enlargement and generalized superficial lymphadenopathy, the rest of the general physical examination showed no abnormality. Examination with a slit lamp by the ophthalmologic consultant revealed many keratitic precipitates and deposits of pigment on the anterior capsule of the lens of each eye. There were numerous clear grayish nodules on the anterior surface of the iris of each eye. Moderate circumcorneal injection was present. The ophthalomologic diagnosis was uveitis due to sarcoidal involvement, a condition which, in combination with the parotid swelling, could be classified as Heerfordt's disease (uveoparotid fever).

Lesions on patient's arm.

Roentgenologic study revealed a mottled density in the lower field of the left lung and bilateral enlargement of the hilar nodes. Reactions to the Mantoux test were negative with dilutions of 1:10, 1:500, 1:1,000, 1:10,000 and 1:20,000. History of a positive cutaneous reaction to a tuberculin test four years previously was later obtained from the patient. Repeated examinations of sputum, gastric washings and cultures revealed no tubercle bacilli. Except for an increase of the sedimentation rate to 47 mm., routine studies of the blood and urine revealed nothing abnormal. The plasma globulin level was slightly elevated. Serologic tests for syphilis were negative, as was the intracutaneous reaction to Frei antigen (lygranum). Microscopic study of cutaneous biopsy specimens by Dr. M. R. Caro revealed moderate hyperkeratosis of the epidermis, with attenuation of the rete pegs. There were focal perivascular accumulations of large mononuclear

cells in the superficial portion of the corium. Sharply circumscribed, rounded tubercles were present in the deeper portion of the corium. These were composed of syncytial masses of lipid-laden epithelioid cells and a few Langhans' giant cells. Lymphocytes were absent, and there were no signs of caseation. There were no changes in the epidermis or superficial part of the corium to account for the clinical appearance of follicular papules, although serial sections of two biopsy specimens were studied. The pathologic diagnosis was Boeck's sarcoid.

Cutaneous lesions persisted unchanged during a two month observation period, which terminated when the patient had an acute schizophrenic episode, which necessitated her transfer to a psychiatric institution.

COMMENT

In classifications of cutaneous tuberculosis such as that proposed by Michelson and Laymon,[7] prognosis has been determined by the clinical features of the eruption, and the results of histologic and immunologic studies have been considered of less importance. The morphologic aspect of the lesion may, however, prove to be an unreliable index of prognosis. On the basis of its clinical appearance, the eruption in the case under discussion was classifiable as a "form which tends to heal relatively rapidly" (lichen scrofulosorum), although laboratory studies, examinations of histologic structure and tuberculin testing suggested a chronic and progressive process. Unquestionably, prognosis for the various types of tuberculids depends to a considerable extent on the resistance of the host to the underlying visceral tuberculosis. Consideration of all the clinical, bacteriologic, immunologic and histologic findings in any given case is necessary for adequate evaluation of the patient's resistance. The relative importance of each of these factors in determining prognosis has not yet been clearly defined, and emphasis on morphology or any other single factor is therefore unjustified. In addition, numerous reports attest the fact that lichen scrofulosorum of typical gross and microscopic morphology may occur in conjunction with other tuberculous lesions, i. e., lupus vulgaris, which have widely different prognostic significances.

SUMMARY

A patient exhibiting lesions clinically typical of lichen scrofulosorum presented histologic and immunologic features of cutaneous sarcoid, as well as sarcoidal involvement of the uveal tract, salivary glands and lungs.

Implications relative to the classification of cutaneous tuberculosis are discussed.

6230 North Kenmore Avenue (40).

7. Michelson, H. E., and Laymon, C. W.: Classification of Tuberculosis of the Skin, Arch. Dermat. & Syph. **52**:108 (Aug.) 1945.

Society Transactions

LOS ANGELES DERMATOLOGICAL SOCIETY

Maximilian E. Obermayer, M.D., *President*

Franklin I. Ball, M.D., *Secretary*

Feb. 11, 1947

Mycosis Fungoides (with Vertebral Involvement, Treated with Nitrogen Mustard). Presented by DR. BEN A. NEWMAN.

R. G., a white woman aged 59, has had a recurrent generalized eczematoid dermatitis for the past eleven years. This condition has been treated with several courses of roentgen radiation. In 1945 a diagnosis of mycosis fungoides was made from a biopsy specimen. During one and one-half years she received roentgen therapy, a course of diphtheria toxoid and injections of antireticular cytotoxic serum without benefit. She was admitted to the hospital again on Nov. 1, 1946. At that time, she presented a generalized dermatitis involving the entire cutaneous surface, consisting of eczematoid infiltration and plaques.

The patient was given two courses of nitrogen mustard (three injections of 6 mg. each) during a two week period. One week after the first injection, involution of the lesions occurred and also pruritus. At the end of three weeks her skin was entirely clear except for a bandlike vesicular patch extending across the right scapula and over the right breast. When the vesicular patch became visible, after the generalized dermatitis disappeared, a diagnosis of herpes zoster was made. Roentgenologic examination of the spine at this time showed a compression of the fourth dorsal vertebra.

The blood cell count, urine and results of the blood chemistry determinations were normal.

Mycosis Fungoides (Treated with Nitrogen Mustard). Presented by DR. BEN A. NEWMAN.

R. C., a white woman aged 58, has had recurrent generalized pruritus for thirty years. For the past three years, she has been under constant care at the Los Angeles County General Hospital and has been refractory to roentgen irradiation for the past year. On Jan. 2, 1947, she was admitted to the Cedars of Lebanon Hospital for nitrogen mustard therapy. At that time, she presented a generalized eruption consisting of bright red, thick, scaly infiltrated patches.

The infiltrate disappeared after three injections of nitrogen mustard, but further therapy was restricted because of a drop in the leukocyte count. The pruritus has decreased considerably, and the infiltrated patches are now pinkish and softer.

Mycosis Fungoides (Treated with Nitrogen Mustard). Presented by DR. BEN A. NEWMAN.

F. S., a white man aged 62, has had a chronic dermatitis of both hands for twelve years. Since the beginning, he has had numerous attacks of a generalized eczematoid dermatitis. In each instance, roentgen irradiation was the only agent that produced involution of the eruption, in spite of considerable topical therapy.

The present attack began three years ago with generalized eczematoid dermatitis. During this period, the patient received repeated courses of low voltage roentgen

irradiation with partial relief, but there were prompt recurrences on discontinuance of therapy. During the past year, he lost about 15 pounds (7 Kg.), chiefly from loss of sleep because of pruritus. He was admitted to the hospital on Jan. 25, 1947, for nitrogen mustard therapy.

Physical examination revealed a generalized eruption consisting of patches and plaques of erythematous eczematoid lesions and erythematous dry infiltrated plaques.

On February 3, treatment with nitrogen mustard was started. He was given four intravenous injections of 6 mg. each in five days. On the sixth day, there was visible involution of the lesions and decrease in the itching. The patient now presents some residual faint pinkish spots on the legs at the site of the former lesions. Histologic examination of the tissue showed the typical polymorphous infiltrate with scattered histiocytes.

DISCUSSION OF CASES OF MYCOSIS FUNGOIDES

DR. L. F. X. WILHELM: I saw a great improvement in the woman who was at the County Hospital for a number of years, but the appearance of her lesions was not altered materially, and when I questioned her about the itching she said that it was still bad. I think that we should see these patients again in thirty or sixty days.

DR. J. R. SCHOLTZ: There appears to be no question that many of the patients treated with nitrogen mustard have been given symptomatic relief and that involution of the skin lesions is substantial. Even though there is no assurance of the permanence of the results, the method certainly affords important palliative treatment in a difficult situation.

DR. A. F. HALL: One patient denied having had any vesicles in the zoster area. This zonal eruption appeared in the form of plaques which were themselves suggestive of mycosis fungoides. It is of considerable interest to conjecture whether this is herpes zoster. If it is, should one assume that it is due to the same virus that produces herpes zoster in otherwise well persons, or is this by any chance a zonal manifestation of mycosis fungoides, or, third, is it just that the eruption takes the form of mycosis fungoides in a person afflicted with that disease?

DR. CLEMENT C. COUNTER: Is the fact that the patient had nausea and vomiting for two days after her injection considered sufficient reason for discontinuing administration of nitrogen mustard, or can it be used again in spite of this reaction?

DR. BEN NEWMAN: In reply to Dr. Hall concerning the zoster-like distribution of the eruption in the first case, this patient had a generalized involvement of the skin with no clear areas except the palms, soles and face. One week following a course of nitrogen mustard—methyl-*bis* (β chloroethyl) amine—therapy, the entire skin was clear except for the wide patches of lesions that are now present. Minute vesicles were scattered among these confluent, erythematous, eczematoid and slightly infiltrated plaques, following the course of the right fourth dorsal nerve. The patient had been complaining of radicular pain in this area for two weeks, as well as generalized itching. When I became aware of this new lesion, a roentgenogram was taken. This showed a destruction of the fourth dorsal vertebra. It has been my impression that this patient has a lymphomatous involvement of the vertebra, with radiculitis and herpes zoster; the zoster reaction in a skin already overwhelmed with mycosis fungoides was soon replaced by the lymphomatous response of the skin. I believe that this lesion is mycosis fungoides, following a radicular distribution.

Nausea and vomiting usually cease after the first two injections. The chief contraindication to further therapy is a significant drop in any of the elements of the blood.

The patients presented this evening are part of a group of 6 patients with mycosis fungoides receiving treatment and under observation at the Cedars of Lebanon Hospital. All the patients were roentgen-ray-resistant, and all have shown a favorable response to nitrogen mustard. The degree of response has varied. At present, my co-workers and I are studying various dosage and interval schedules of therapy. I do not believe that nitrogen mustard will cure this disease, but it has already provided immeasurable relief to some patients. With a chemical capable of destroying certain types of neoplastic cells, even temporarily, it is probable that we may be heralding an era of a new method of management (chemotherapeutic) of malignant disease.

Darier-Roussy Sarcoid (Gumma?). Presented by DR. STANLEY O. CHAMBERS and DR. STANLEY C. ANDERSON.

Granuloma Annulare of the Arms and Face. Presented by DR. MOLLEURUS COUPERUS.

Radiodermatitis with Ulceration (Successfully Treated with Radon Ointment). Presented by DR. JUD R. SCHOLTZ.

Acrosclerosis (Favorable Response of Ulcers to Neostigmine Hydrobromide). Presented by DR. MOLLEURUS COUPERUS.

DISCUSSION

DR. NELSON PAUL ANDERSON: I am treating a patient with Reynaud's syndrome and sclerodactylia who has had ulcerations of the finger tips. He weighs about 210 pounds (95 Kg.) and is 6 feet 2 inches (188 cm.) tall. We have given him five injections of tetraethylammonium bromide. It is apparently a potent drug, because shortly after the injection, which is given very slowly, he can feel increased warmth in the hands and feet and is able to move them with more freedom. Just what the ultimate outcome will be, I cannot say, but I do feel that the treatment may at least lead to some useful information. It may possibly give an indication as to which are the favorable cases for sympathectomy. I doubt that this drug attacks the fundamental perivascular system itself. In the October 1946 issue of *Surgery* there is an excellent article on this drug. I warn you not to administer the drug when in a hurry, because it is necessary to spend an hour or two with the patient.

A Case for Diagnosis (Carcinoma? Pseudoepitheliomatous Hyperplasia Associated with Infectious Mononucleosis and Primary Tuberculous Complex?). Presented by DR. J. WALTER WILSON.

Psoriasis (Hypertrophic Lichen Planus?). Presented by DR. NELSON PAUL ANDERSON.

A Case for Diagnosis (Lupus Erythematosus?). Presented by DR. H. C. L. LINDSAY.

A Case for Diagnosis (Pigmentation of All Finger Tips from Unknown Extraneous Source, Factitious). Presented by DR. J. WALTER WILSON.

A Case for Diagnosis (Dermatitis Vegetans?). Presented by DR. SAMUEL AYRES JR.

A Case for Diagnosis (Rhinophyma-like Eruption of Face? Lichenoid Eruption of Trunk?). Presented by DR. SAMUEL AYRES JR.

A Case for Diagnosis (Carcinoma of the Lip? Lupus Erythematosus?). Presented by DR. THOMAS W. NISBET.

A Case for Diagnosis (Erythema Induratum? Nodular Vasculitis?). Presented by DR. NELSON PAUL ANDERSON.

Maximilian Obermayer, M.D., *President*

Franklin I. Ball, M.D., *Secretary*

March 11, 1947

Porokeratosis of Mibelli. Presented by DR. SAMUEL AYRES JR.

N. A., a white boy aged 12, presents a lesion on the right side of the neck, approximately the size of a nickel with a narrow, elevated border which appears to be made up of individual elements showing dry, hyperkeratotic scaling along the top of the border. The lesion developed about nine months ago. There is one small pinhead-sized nodule within this circle, and there is a slightly lichenified appearance on the surface of the area enclosed by the ring.

Histologic examination reveals that the most characteristic feature is in the epithelial layer, the central portion of which shows hyperkeratosis and parakeratosis with a follicle filled with keratin. The prickle cell layer immediately surrounding the follicle is acanthotic, giving the appearance of a ridge with a furrow. The basal cell layer is intact, with some perivascular infiltrate in the upper cutis.

DISCUSSION

DR. KENNETH L. STOUT: Clinically, I thought this was granuloma annulare, and when I looked at the section I found nothing to change my mind.

DR. L. H. WINER: I agree clinically with the diagnosis, but the histologic section did not show porokeratosis of Mibelli. Of course, every section of porokeratosis of Mibelli would not necessarily show the characteristic histologic appearance. Of interest in the slide are the porokeratotic scales. These have been reported adjacent to the hyperkeratotic disks of porokeratosis of Mibelli and are located at the periphery of the disks of porokeratosis. However, the infiltration of the cutis was deep and stellate in arrangement. I could not see any collagenous necrosis. I would suggest another biopsy.

DR. NELSON PAUL ANDERSON: I think the slide is fairly typical of porokeratosis of Mibelli. I think a good deal of variation is seen, depending on (1) the sites of biopsy and (2) where the actual slide is sectioned. I happen to have Mibelli's original article on porokeratosis, and I am sure that from a number of photomicrographs in this article one would accept the section as typical.

DR. SAMUEL AYRES JR.: My experience with this disease has been limited to 1 case, and in that instance total destruction was the only method that appeared to be successful—either excision or destruction, with electrodesiccation and curettage. I think this is the method of choice.

DR. NELSON PAUL ANDERSON: In Cleveland, at the last meeting of the Academy, Dr. Cole showed a patient who had perhaps fifty lesions of porokeratosis of Mibelli around the face and neck. Under moderate vitamin A dosage, 25,000 units three times a day, there had been at least a 75 per cent regression, according to the photographs and the condition of the patient. Dr. Allen in San Diego has a whole family with porokeratosis of Mibelli, with one patient who had ten to twelve lesions on the neck, and there has been dramatic improvement with vitamin A therapy.

Chronic Urticaria of Seventeen Years' Duration. Presented by DR. NELSON PAUL ANDERSON.

Tinea Capitis Favosa in a Girl Aged Eleven Years, a Native of the Island of Rhodes. Presented by DR. M. E. OBERMAYER and DR. J. WALTER WILSON.

Favus of the Scalp. Presented by DR. HAROLD E. ANDERSON and DR. J. WALTER WILSON.

M. K., a white woman aged 28, has had a fungus infection of the scalp continuously since infancy. Nine siblings and two paternal aunts are said to have been similarly infected, in some of whom the infection is still present. When the patient was 6, the entire family was treated at the Mayo Clinic with roentgen epilation; some were cured. This patient states that such epilation was carried out three times in her case. Many other attempts have been made at treatment, with uniform lack of success.

Examination under Wood's light revealed a dull greenish fluorescence extending along numerous hair shafts for 2 to 3 cm. from the scalp. Potassium hydroxide preparations of such hairs showed hyphal threads within as well as around the hair shaft, together with chains of spores and occasional air bubbles. Culture was obtained on Sabouraud's glucose agar, but only on the third attempt because of secondary involvement with bacterial and nonpathogenic fungi. Sufficient growth has been obtained, however, to enable microscopic diagnosis of Trichophyton schoenleini to be made.

No treatment has been instituted to date.

DISCUSSION OF TWO PRECEDING CASES

DR. H. P. JACOBSON: I saw only 1 of the 2 cases. The scalp lesions in that patient consisted of several roughly circular patches of inflammatory alopecia with some scarring but no scutula. The culture is characteristic. The presenter states that topical treatment has proved of no avail. My therapeutic suggestion would be topical employment of 15 per cent thymol in oil of thyme. The scalp should be shaved once a week, and the thymol application should be put on once daily with an applicator. I have had success in the management of tinea capitis (microsporic and trichophytic) with this fungicidal agent.

DR. L. H. WINER: The first patient did not have scutula but did have the typical atrophic scars of favus. In the second case scutula were present, and on being daubed with alcohol they became yellow, which is a characteristic of favus.

DR. H. C. L. LINDSAY: In 1913 Dr. Sabouraud showed some patients who had had favus since their earliest days. He stated that he had treated the parents of these patients for favus and, furthermore, that in a study of the records of St. Louis Hospital, Paris, it was possible to prove that favus had been present in the ancestors of these patients for generations. At this time Dr. Sabouraud was epilating the scalp of patients with favus with roentgen rays as a preliminary treatment. Success in treatment of favus among illiterate persons is most difficult. Fortunately, hairs in which favus is growing are not so brittle as when they are invaded by small-spored ringworm. Thus favus hairs can be plucked manually. Favus was not infrequently seen at St. Louis Hospital, and a considerable number of the patients had permanent scars and alopecia in patches.

DR. CLEMENT E. COUNTER: Is the temporary epilation of the scalp expected to cure favus? One of these patients presented tonight has had epilation three times. She still has the disease.

DR. BEN NEWMAN: In reference to temporary roentgen ray depilation, our attention should be directed to two important considerations: one, that in the past number of weeks approximately a dozen cases of Microsporum audouini infection of the scalp have been uncovered in and around Los Angeles and that we may expect an epidemic form of the disease, if the same pattern is followed here as has occurred in all the larger cities throughout the county; two, that heretofore it has not been necessary, nor has it been the custom, to employ roentgen ray depilation in the treatment of tinea capitis in this area. Hence, if we desire to control the spread of this epidemic, we must recognize the need for such depilation and establish this procedure as a proper and customary type of therapy in this community to avoid medicolegal complications. Certainly, the merits and need for depilation in Microsporon audouini infections of the scalp require no discussion among dermatologists.

DR. H. C. L. LINDSAY: A few years ago efforts to demonstrate favus fungus in hairs by stain were not always successful. Hot carbolfuchsin was used to stain, and dilute nitric acid was used to decolorize. Supposedly the decolorizing of the fungi should be slower than that of the other stained material. Unfortunately, the hair would decolorize at approximately the same speed. Accidentally, I placed some partially decolorized stained hairs containing favus in a watch-glass container which was partially filled with aniline oil. I forgot about these hairs for a week. When I examined them with the microscope, the fungus in certain parts could be seen retaining the dye, while the rest of the field was clear.

DR. NELSON PAUL ANDERSON: No one has ever seen favus develop in an adult. If complete epilation had been carried out on this woman and the disease cured, she would not have been reinfected when an adult. Sabouraud said he had never seen a case of favus transferred to an adult. I am convinced that certain types of favus are curable by manual epilation. I have had 3 cases of favus, 1 involving the thumbnail, 1 involving the scalp of a little girl and 1 involving the scalp of the aunt of the little girl. The mother faithfully epilated all the hairs from the scalp of the daughter and faithfully scraped her own thumb nail. Both patients are now well. The scalp of the maternal aunt still presents widespread involvement.

DR. J. WALTER WILSON: I am glad that Dr. Obermayer and Dr. Anderson afforded me the opportunity of carrying out the mycologic studies in these 2 cases, which I believe are representative of favus under treatment and scrupulous cleanliness. Growth of T. schoenleini was obtained only with difficulty from the adult because of the presence of other nonpathogenic fungi, which repeatedly suppressed

its growth. The dull greenish fluorescence extending for 2 or 3 cm. distally along the hair shaft is typical. The adult is able to trace the disease through her father to his parents, who came from Luxembourg; the child's parents came from the Island of Rhodes.

DR. HAROLD E. ANDERSON: My patient, the adult in the second case, had much heavy, dry scaling extending along and matting together the hair shafts when I first saw her. It reminded me of what is called tinea amiantacea. A microscopic examination of potassium hydroxide preparation of hairs and scales selected for study revealed numerous mycelia and spores. Secondary invaders overgrew the culture mediums, but Dr. Wilson was finally able to isolate T. schoenleini. The familial tendency, the clinical appearance and the cultural study all are typical of tinea favosa. She has already had roentgen ray epilation at least three times, once at the Mayo Clinic and once at the University of Iowa. She was treated at the Mayo Clinic in 1924. A direct microscopic examination of hairs at that time revealed fungi, but the result of the cultural study was not reported. The postepilation therapy was carried out at home in Iowa, and apparently the parents were not diligent enough with this therapy.

Basal Cell Epithelioma (Morphea Type). Presented by DR. SAMUEL AYRES JR. and DR. JOSEPH I. MIROVICH.

DISCUSSION

DR. A. F. HALL: In several cases in which I have tried treatment by radiation the condition has proved recalcitrant; I think the lesion has to be destroyed in some other way, such as by cautery.

DR. ANKER K. JENSEN: At the risk of repetition, I would again say that in the management of cutaneous malignant growth we must carry out the radical measures first. Conservative management is at no time justified in the handling of cutaneous neoplastic disease. This applies both to radiation therapy and to surgical procedures. I confess partiality to cautery surgery, but of course I am also employing radiation. My radiation dosages are much heavier than those employed in the present case. I am inclined to agree with Dr. Hall that the epithelioma in our present patient has the aspects of a morphea-like lesion, and this could possibly be a factor in its apparent radioresistance. There is, of course, little if any danger of distant metastasis from this lesion, it being a basal cell growth. The clinical picture at present, however, indicates a steady but progressive extension and activity, and unless eradicated completely it will probably result in a rodent ulcer. I recommend radical surgical treatment by means of cautery.

DR. L. H. WINER: Basal cell carcinomas of the scalp are occasionally of sebaceous gland origin and as such are radioresistant. I agree with the preceding discussers that excision followed by skin graft is the treatment method of choice.

Blastomycosis of the Foot (North American). Presented by DR. W. H. GOECKERMAN, DR. L. F. X. WILHELM and DR. MOLLEURUS COUPERUS.

Acanthosis Nigricans. Presented by DR. NELSON PAUL ANDERSON.

J. H. D., a white youth aged 18, had a pinkish eruption under the arms, on the sides of the trunk and on the abdomen about three years ago. Physical examination at the present time reveals a rough, elevated, dirty brownish, reticu-

lated, papular hyperkeratotic eruption about the center of the trunk, on the anterior and posterior axillary folds and in the pubic region. There is also involvement about the inguinal regions and the lower part of the abdomen and in the bends of the elbows. The center of the chest and back and the sides of the neck are clear.

Histologic examination of the tissue showed a verrucous acanthotic process, with one area where there were four nests of cells just beneath the epidermis, like a nest of melanoma cells. Many of the epidermal cells were vacuolated.

Treatment has consisted of administration of massive doses of vitamin A, with great improvement. The patient has recently been using sodium thiosulfate soaks at the suggestion of Dr. E. S. Lain, of Oklahoma City.

DISCUSSION

DR. L. H. WINER: My clinical impression was the close resemblance of this case to parapsoriasis, especially in regard to the lesions on the abdomen and flanks. However, the verrucous lesions in the right axilla were characteristic clinically of acanthosis nigricans, as was the histologic section. Histologically, one cannot differentiate the benign form from the malignant form of acanthosis nigricans.

Urticaria Pigmentosa. Presented by DR. STANLEY O. CHAMBERS and DR. STANLEY C. ANDERSON.

Verruca Plana of the Face Treated by Posthypnotic Suggestion. Presented by DR. MAXIMILIAN E. OBERMAYER.

D. P., an unmarried woman aged 21, noticed the appearance of a group of flat warts on the right side of her chin two years ago. The dermatologist who was treating her at that time with fractional doses of roentgen rays for acne vulgaris intimated that the warts would probably disappear with continuation of treatment. However, the verrucae spread rapidly, and when the course of roentgen therapy had been completed they involved most of the face. Various dermatologists removed the ordinary warts which appeared on the fingers of the right hand by means of electrodesiccation. Injections of bismuth subsalicylate in oil, given over a period of eight months, as well as subsequent applications of peeling ointments and solid carbon dioxide and the use of lotions in combination with ultraviolet irradiation, had no effect on the verrucae planae of the face.

When I saw the patient for the first time, on Dec. 5, 1946, her face was studded with verrucae of the flat juvenile type and lesions were also present on the forehead, the anterior portion of the scalp and the sides of the neck. Most of the right side of the face and neck was covered with confluent plaques, while the lesions on the left side were more scattered.

When it became clear that the oral administration of bismuth (sobisminol mass) and the local use of a 20 per cent solution of podophyllin in acetone had failed to benefit the patient, her mother was told that I had no other therapeutic suggestions, and the irrational behavior of warts was discussed and illustrated by an account of Dr. Bloch's experiments in Zurich. A few days after this discussion, the mother suggested that arrangements be made for treatment by posthypnotic suggestion. I agreed. In order to complete my record of the case, biopsy of a lesion was performed. The section showed the features typical of verruca plana juvenilis.

The patient was then referred to Dr. Ralph R. Greenson, certified by the American Board of Psychiatry. She saw Dr. Greenson only three times, at weekly intervals. Two weeks after the last consultation, in accordance with a suggestion made during light hypnotic sleep, all verrucae had disappeared.

The skin today reveals no trace of the lesions.

DISCUSSION

DR. A. F. HALL: I think we will all admit that the warts either went away spontaneously or were cured by suggestion; to believe the former demands too much of coincidence. I think we will admit that Rasputin, the "black monk of Russia," controlled the czarevitch's hemophilia by suggestion. I think most of us, if not all, can cite examples of warts that have been cured by prayer or wishing or out and out suggestion, either in our childhood or in our practice; since Bloch put this type of treatment on a respectable basis, I expect that "suggestion" is the proper word. It is intriguing to conjecture regarding the mechanism involved. I remember seeing an article on the subject by Karl Zwick in the ARCHIVES (Zwick, K.: Hygiogenesis of Warts Disappearing Without Topical Medication, *Arch. Dermat. & Syph.* **25**:508 [March] 1932). The author's conjecture is that the treatment of warts by magic and suggestion often includes traumatism of the wart, e.g., by rubbing it with a potato or a penny or an apple. He also mentions the cure of warts by suggestion without touching the wart. He feels that the factor which the two types of approach have in common is perhaps that the wart is unfavorably affected by tissue juices; he advances this theory, because in the inoculation of warts, if any of the innoculum gets into the subcutaneous tissue, a new wart will not appear. He further points out that, by psychic methods, extravasation of serum and diapedesis of blood cells can take place under hypnosis. He thinks it is through some such mechanism as this that warts may be influenced. Were I to "take it from there," I should hypothecate that if there is a tissue fluid or some substance in the corium inimical to warts, it may be the same substance that makes the difference between the bleeding of ordinary tissue and the bleeding of a wart. The prolonged bleeding time, seen when a wart is cut, may be due to the absence of thromboplastin in warty tissue; it may be this substance, present in the corium and absent in warts, that is inimical to the "life" of a wart. One might try injection of thromboplastin into the base of a wart as treatment, in circumstances which would eliminate the factor of suggestion.

DR. J. R. SCHOLTZ: In my opinion, most of the treatments that we use for warts act by suggestion, and as far as I am concerned, treatments which are harmless and do not leave scars are the most desirable. I can see no therapeutic sense in using any method which causes a scar in place of a benign, harmless lesion. There is no doubt that psychotherapeutic methods are effective in the treatment of warts. Recently in the clinic of the Los Angeles County Hospital, a boy aged 8 years was seen with four to six large verrucae vulgares on his hands. They were bright blue, and it was found that he had been treating them daily with an indelible pencil. We encouraged him to continue and stated that the warts would probably disappear in about three weeks. On his return a month later, all the warts were gone.

DR. MAXIMILIAN OBERMAYER: There are two reasons for this presentation. First, I believe that dermatologists should become better acquainted with the extraordinary fact that an infectious disorder may be amenable to suggestive therapy. Obviously, first hand knowledge of the data in an authenticated case

will be much more enlightening than information obtained by hearsay or from an unassimilated skimming of psychiatric literature, as the late Dr. Bloch fully realized when he conducted his now famous clinical experiment with Swiss school children. Second, I believe that speculation about the nature of the working mechanism underlying the indisputable results of suggestive therapy will prove of value. In my opinion, it is most rational to assume that the neurovascular mechanism is involved. The psychodynamic factors which lead to contraction of the capillary vessels and consequent pallor in certain emotional states are perfectly familiar. Logically one may assume, then, that suggestion can produce a similar spasm of the capillaries, with consequent shutting off of the blood supply to the lesions. Dr. Greenson approached the problem with such an assumption in mind.

Since a preliminary interview revealed no psychoneurotic features, the psychiatrist expressed the belief that while the treatment could produce no ill effects, its chance of success was small. At the end of the first interview, the patient was told to imagine for a few minutes before bedtime each night that her face was covered by cold compresses and was beginning to itch and tingle. At her second visit, she gave the encouraging report that she had had the sensation of itching. An attempt at hypnosis was followed only by a state of deep relaxation without sleep, during which it was suggested that her face would feel cold, her skin would itch and the warts would begin to fall off. The patient began the third and last consultation by stating that the warts had commenced to become scaly. Once more hypnosis was attempted, and a state of light sleep was produced. It was then suggested that her face would feel cold and turn pale and that the lesions would itch and fall off within two weeks. The patient was awakened and told that no further interviews were contemplated because her warts would shortly disappear. Two weeks later her skin was clear of all lesions.

I should be the last to propose that posthypnotic suggestion be employed routinely in the treatment of verrucae. On the other hand, since the shortcomings and unreliability of all the so-called approved methods of treating warts are well known, I believe that suggestive therapy should be given a trial when conventional methods have proved useless and extensive involvement has raised a problem from the cosmetic standpoint, as in this case. That such therapy will succeed with only a limited number of patients is obvious and that it should be carried out only by a fully trained psychiatrist well versed in the technic and aware of the dangers of hypnotic suggestion is equally clear. Certainly most dermatologists would willingly accept a dozen failures with psychotherapy rather than invite a single instance of the dermatitis actinica producd by roentgen therapy. The sooner suggestive therapy is given a fair and unbiased trial by the dermatologic profession the sooner its limitations and suitability will be established.

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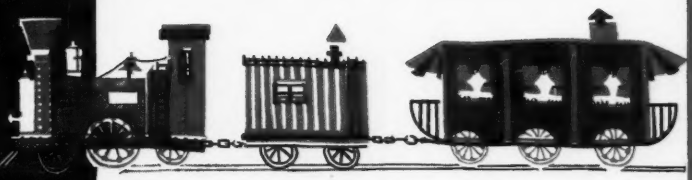

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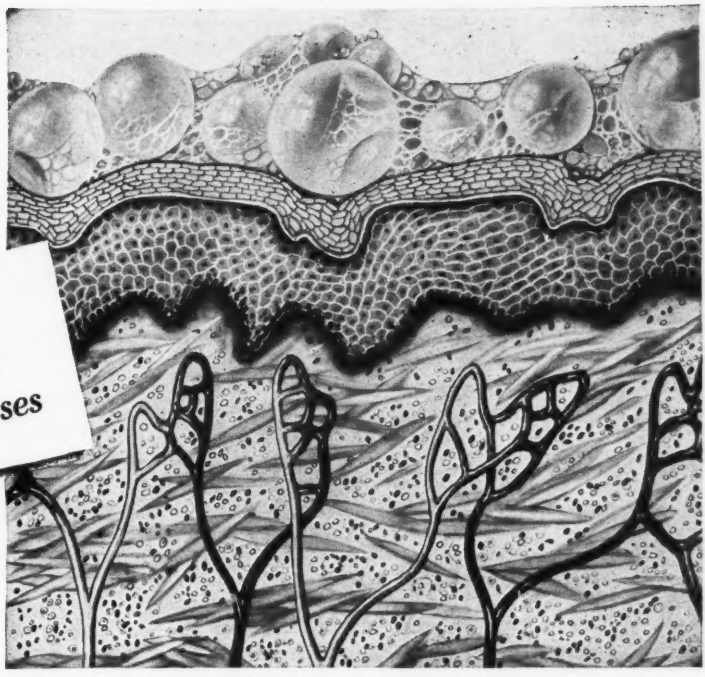

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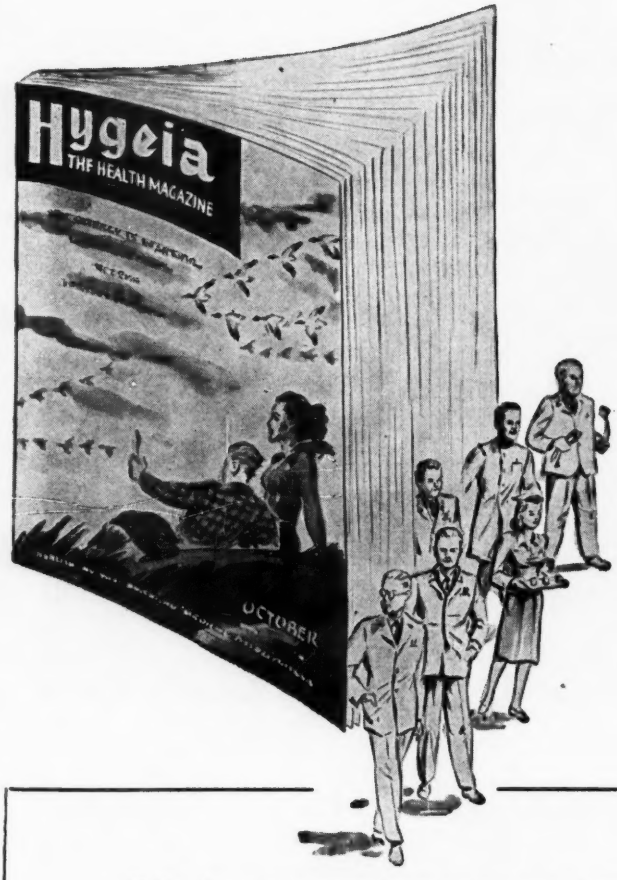

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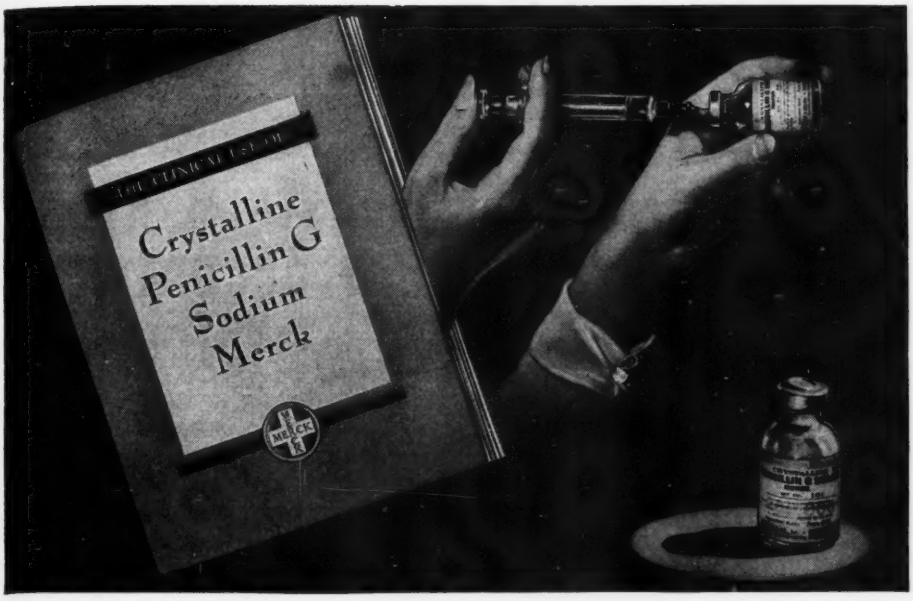

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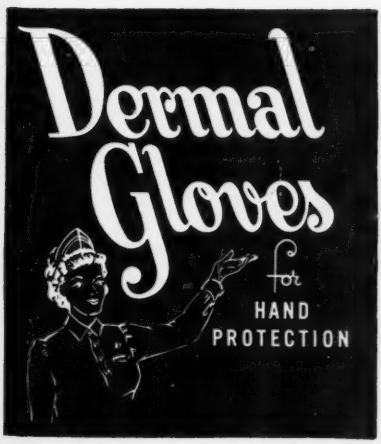

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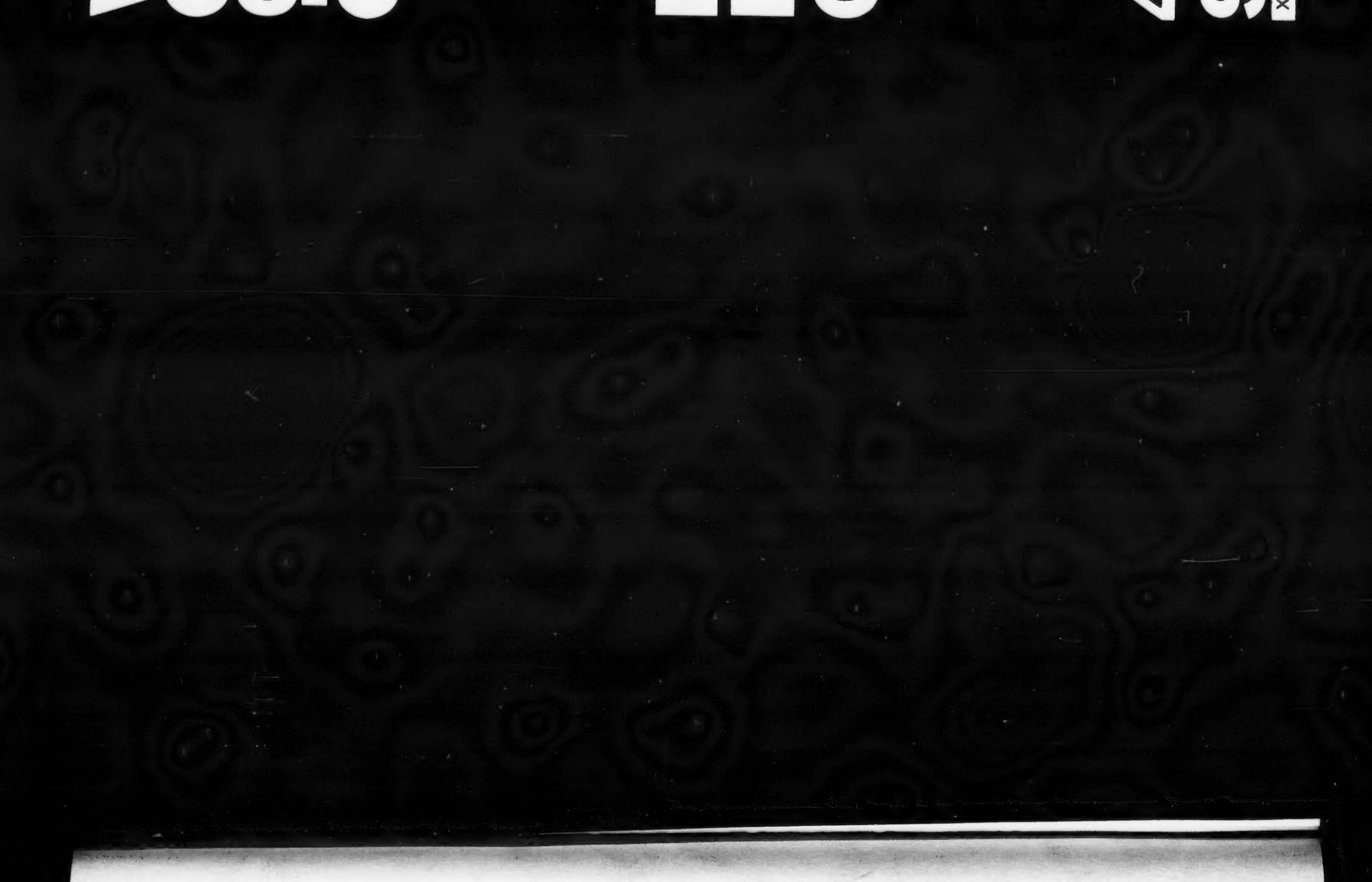
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